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TRANSACTIONS

OF THE

American Climatological and Clinical Association

FOR THE YEAR 1923

VOLUME XXXIX

"The object of this Association shall be the study of Disease especially of the Respiratory and Circulatory Organs and of Climatology and Hydrology."—*Constitution.*

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1923

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The Association assumes no responsibility for the statements and opinions expressed in the papers read at its meetings.

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1923

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Beverley Robinson	1899
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Samuel A. Fisk	1902
Norman Bridge	1903
James C. Wilson	1904
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E. L. Shurly	1906
Thomas Darlington	1907
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1887. †Smith, Fremont, Winter Park, Fla.

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1898. Williams, Harold, 528 Beacon St., Boston, Mass.

---

†Died 1922.

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†Died 1923.

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Wood, Nathaniel K., 520 Beacon St., Boston, Mass.

MINUTES.

The Fortieth Annual Meeting of the American Climatological and Clinical Association was held at the Clifton House, Niagara Falls, on May 23rd and 24th, 1923. Fifty-seven members were present, and telegrams and letters were received from thirty-three others expressing their regret at their inability to be present. A number of guests also were present from Buffalo, Niagara, and Toronto.

Under the skillful direction of Dr. Richardson, President, the meetings were held in five sessions, three on the first day with full attendance at all of them, and two sessions on the second day were sufficient to complete the program as a number of the readers found at the very last minute that they could not leave home. There was much interest in the program and a free discussion took place such as is possible in a small hotel with the meeting practically confined to members of the Association.

Thursday afternoon, at the invitation of Sir Adam Beck, a large number of the members and the ladies visited the Hydro-Electric Installation Plant now nearing completion.

At the annual dinner on Thursday evening, Dr. Richardson presided, at which forty-eight members were present. Dr. Porter of Toronto, on behalf of the Canadian hosts, was the principle speaker at the dinner. Among the other speakers were Dr. Judson Daland, Dr. Thomas Coleman, Dr. Paul Ringer, and Dr. Jeremiah Metzger. On behalf of the members of the Association, Dr. Darlington presented Dr. Babcock with a beautiful gold headed cane. This meeting was the thirtieth anniversary of Dr. Babcock's election to the Association, and it has been suggested that some recognition be taken of this fact and of his unremitting interest and attendance. The inscription on the cane reads as follows: "Presented to Robert H. Babcock, M.D. with love and esteem of his colleagues in the American Climatological and Clinical Association at the Niagara meeting,

1923." Dr. Babcock made a brief response, appreciative of the good wishes of the members of the Association, as expressed in their gift.

At the same time the annual dinner was being held, Mrs. Richardson entertained the ladies of the party at dinner, and later they joined the men to listen to the speaking. The Glee Club sang with vigor and after they had sung for the ladies' party, Mrs. Dunn in turn sang for the Association.

Early Friday morning one group of the members went to visit the Diabetic Clinic of Drs. Banting and Best in Toronto, while another group visited Perrysburg to see the heliotherapy work being done there under Dr. Lo Grasso.

Business.

The business session of the first day was confined to the routine reports, the passage of the amendment to the constitution whereby the Council was increased from five to seven members, appointment of the necessary committees, etc. The Nominating Committee was as follows: Dr. Lawrason Brown, Dr. C. E. Edson, and Dr. John B. Hawes, 2nd.

At the annual meeting on Thursday, the Nominating Committee reported the following officers:

President, Dr. Gordon Wilson.
Vice-Presidents, Dr. H. R. M. Landis and Dr. A. M. Forster.
Secretary and Treasurer, Dr. A. K. Stone
Recorder, Dr. Cleaveland Floyd.

The Council for this year consists of the following:

Lawrason Brown
Carroll E. Edson
Wm. Duffield Robinson
Charles W. Richardson
Gerald Webb
Lawrence Litchfield
J. Woods Price

Delegate to Congress of the American Physicians and Surgeons: Dr. Thomas Darlington; Alternate, Dr. Walter L. Niles.

The ballot was then taken for new members and the following were elected:

Honorary membership: Prof. A. Rollier of Switzerland.
Active membership:

Thomas C. Kelley, Philadelphia, Pa.
J. L. Moorman, Oklahoma City, Okla.
John W. Finn, Prescott, Arizona.
Herbert M. Rich, Detroit, Mich.

The following members of the Association have died during the year: Life members Frank Fremont-Smith and Walter B. Platt; and William M. Hart who was elected to membership last year.

The following list of names were presented for membership:

Earl Sprague Bullock, Silver City, New Mexico.
Nathan Barlow, U.P.H.S., New Haven, Conn.
Alfred H. W. Caulfield, Toronto, Canada.
Chas. W. McGavran, Columbus, Ohio.
Frank Porter Miller, Los Angeles, Cal.
Maurice C. Pincoffs, Baltimore, Md.
Clayton W. Greene, Buffalo, N. Y.
Byron B. Brown, Buffalo, N. Y.
Allen A. Jones, Buffalo, N. Y.
Horace LoGrasso, Perrysburg, N. Y.

The above list of names in addition to those already presented at previous meetings makes a waiting list of about twenty names. There are only two or three vacancies to bring the membership of the Association up to its full number. It is therefore of the greatest importance that the members should give to the Council their opinion of these men, so as to enable the Council at its next meeting to make the wisest selection from this wealth of material.

The Treasurer stated that on account of the publication of two volumes of Transactions during the past year, at a cost of $1,938 there remained in the treasury only funds enough to carry out the ordinary expenses of the society. On motion by Dr. Wood, it was voted that a special assessment of $5.00 be levied to enable the Recorder to publish the papers of this meeting as soon as possible.

In the discussion of the next place of meeting, the attractions of Pittsburgh, together with an earnest invitation to visit there was extended by Dr. Litchfield, and seconded by several others. Members presented invitations to visit Cincinnati, Asheville, and Atlantic City. The matter was finally left to the discretion of the Council.

Vote of thanks were given to the President for the admirable way in which he conducted the meetings, and to Sir Adam Beck for the courtesy extended by him in the visit to the Hydro-Electric Plant. Dr. Gordon Wilson made a brief speech of appreciation of the honor of his election as President.

MEMORIAL.

FRANK FREMONT-SMITH.

Presented by CHARLES W. RICHARDSON.

Frank Fremont-Smith was born in Hillsboro, New Hampshire in 1856. He attended the schools in his native state. He taught in the public schools a year before entering Dartmouth College. He graduated from Dartmouth in 1880. He took his first medical year at Ann Arbor, University of Michigan, and then transferred to the University of Pennsylvania from which he graduated in 1883. He entered active practice in St. Augustine, Florida. In 1891 he opened a summer practice in Bar Harbor, Maine, which he maintained until his death.

In 1898 he moved his winter practice to Palm Beach, and in 1905 to Washington, maintaining the latter as his home until 1915.

In 1890 he journeyed to Berlin where he took up work with Koch. In 1891, he published a paper, the result of his study in Berlin, entitled, "Radical changes in the Application of Koch's Lymph," Medical Record, October 17, 1891.

Among his written papers we may mention the following:

Pylonephritis—N. Y. Med. Journal, Dec. 1906.

Local Arterio-sclerosis—J. A .M. A., March 7, 1908.

Arterio-sclerosis of the Young—Amer. Journal of the Med. Sc., Feb., 1908.

The Effect of Florida Climate upon Acute and Chronic Diseases —Boston Med. & Surgical Journ., Dec., 1908.

Treatment of Diabetes—Boston Med. & Surg. Journ., 1916.

Dr. Fremont-Smith was a member of the Maine Medical Association, formerly associate professor of Pediatrics, George Washington University; at one time physician to the Hospital for Foundlings and the Garfield Hospital, Washington, D. C.

For a number of years he was an active member of this association, keenly interested in its work. Well trained in medicine he soon became an accurate diagnostician and early attracted the attention of the profession and the laity. He built up quite an extensive practice both at Palm Beach and Bar Harbor.

His last visit to the writer was made the fall before his death, when on his way to Winter Park, where he had established himself for the winter. At this time he seemed to have regained his old buoyancy and looked forward with expectations to a successful new start in Florida. We all knew him to be a courteous and kindly member, with delightful manner and the dignity that becomes a physician.

He passed away at Bar Harbor, September 29, 1922.

LIEUTENANT-COLONEL WILLIAM MALLOCH HART, M.C.

Presented by CHARLES D. PARFITT.

William Malloch Hart was born at Winnipeg on April 27th, 1881, the son of the Rev. Thomas Hart, D.D., and of Isabella Margaret Malloch Hart. His father in the late seventies had gone to Manitoba to organize missions and to help in the founding of educational work. For thirty-eight years he was Professor of Classics in Manitoba College. Mrs. Hart is the daughter of the late Judge John Glass Malloch of Perth, Antario.

Hart graduated in Arts and Medicine from the University of Manitoba, taking his medical degree in 1907. In the autumn of that year he entered the Adirondack Cottage Sanitarium as a patient and a year later became assistant physician. Afterwards he worked at the Gaylord Farm Sanatorium and left there in 1910 for Duluth to become the executive secretary of the County of St. Louis Tuberculosis Commission. Here he organized anti-tuberculosis work on broad lines and Nopeming Sanatorium was built under his direction. He became its first Superintendent and was given the privilege of nominating his successor. His methods and example have been a constant inspiration toward the right kind of work.

Late in 1911 Dr. Hart left Duluth to undertake further pioneer work in Saskatchewan, where he organized an effectve campaign and

had the satisfaction of seeing the large, well-planned Saskatchewan Sanatorium nearing completion when war was declared. While prosecuting his campaign during these five pioneer years of hard physical work, often greatly discouraged, he wrote freely for the education of both the medical profession and the public.

With the outbreak of war on August 4th, 1914, he immediately applied for a commission as a combatant and then in the Army Medical Service. With no commission immediately in sight he enlisted on August 8th as a private in a mounted company of the Corps of Guides. His wish was to become a combatant as an atonement for his previous pacifism. At Valcartier, where he was now sergeant, he was transferred to the Machine Gun Section of the 5th Battalion, 2nd Infantry Brigade, which went overseas with the first contingent. Late in November at Salisbury Plain he learned that on August 4th he had been appointed Provisional Lieutenant of the A.M.C. Early in January, 1915, he was appointed Medical Officer to his old Battalion, which, in February, joined the B.E.F. in Belgium.

In April, 1915, at a Regimental Aid Post, far advanced in the Yprès salient, he went through the second battle of Yprès, where gas was first used. His Aid Post, filled with about eighteen severely wounded who could not be moved, was isolated for some days, and Captain Hart with his one remaining helper ventured forth to obtain ambulances. The ambulances could not be got through and Hart was obliged to attempt the return journey of three miles to his wounded, alone, and in broad daylight. He was taken prisoner on this journey, April 27th, the sixth day of the attack on that part of the line. Professor J. G. Adami in "The War Story of the C.A.M.C." quotes freely from Hart's diaries, describing this thrilling episode. When exchanges were made two months later he was the first Canadian to be returned to England. In the autumn he returned to the front with the 3rd Canadian Field Ambulance, with which he spent fifteen months in Belgium and on the Somme. His modest diaries show his characteristic intense devotion to duty. While in France he was twice mentioned in despatches, promoted to the rank of temporary Major, and awarded the Military Cross. This he received at Buckingham Palace in March, 1917.

Late in 1916 Major Hart was transferred to England, and, after working at different military hospitals, was, because of his special qualifications, appointed O. C. of the Canadian Special Hospital for Tuberculous Soldiers at Lenham, in September, 1917. In May, 1918, he was promoted to the rank of Acting Lieutenant-Colonel and in January, 1919, he became temporary Lieutenant-Colonel. This special hospital Lieutenant-Colonel Hart carried through from start to finish. He returned to Canada in October and was demobilized in January, 1920.

Hart was then appointed by the Department of Soldiers' Civil Re-establishment to a Board of Consultants to consider all phases of the condition of the tuberculous soldier, his treatment and re-establishment. After an extensive survey he contributed largely to the various reports, and himself wrote the special report relating to the care and employment of the ex-service man after leaving the sanatorium. This timely and exhaustive review of after-care problems in general contained many practical recommendations. He was retained at Ottawa as a permanent consultant and advisor with regard to the pensioning and re-establishment of tuberculous soldiers. For this work Dr. Hart was eminently suited, having ample knowledge of conditions and the sympathy that comes from comradeship in arms. In all his work he displayed the most complete unselfishness and devotion. Moreover, he was methodical, untiring, judicial, fair, and, above all, kind. A confrère writes: "Surely the interests of ex-service men were never better served than in his careful, studied, sympathetic and just decision."

This exacting work weighed heavily upon him and he became so worn physically that, following an operation for appendicitis, probably long present, he died April 17th, 1923, in his 42nd year. He was buried at Winnipeg with full military honors.

He leaves one daughter, Mary Dorothy Malloch Hart, and his wife, Barbara Beatrice Hart.

To him, approaching death was "An interesting experience, which, while not entirely agreeable is by no means unpleasant—from which there is nothing to fear—nothing to be afraid of—rather like taking a strange anesthetic."

With the handicap of a fragile body and later of definite disease

he carried through cheerfully the work of a strong man. The war had interrupted a promising medical career and army life became a dominating interest, but he hoped still, when the service for which he was so peculiarly fitted made less demands, to return to internal medicine.

His winning personality, sincerity, fine sense of honor, and keen sense of humor endeared him to his friends. He had a remarkable gift for friendship and a wonderfully fine touch in all his contacts.

It was on the very threshold of his membership to this society that Lieutenant-Colonel "Billy" Hart passed from us, so he was known to only very few of the members; but with those who knew him he had gone far beyond into the inner places of their hearts and lives.

WALTER BREWSTER PLATT, M.D.

Presented by Louis V. Hammon.

Dr. Walter Brewster Platt, a member of this association since 1887, died at Baltimore on October thirtieth, nineteen hundred and twenty-two of heart disease.

Dr. Platt was born at Waterbury, Connecticut on December twentieth, eighteen hundred and fifty-three. He was the son of Gideon Lucian and Caroline (Tudar) Platt. His father was a prominent physician and surgeon, at one time President of the Connecticut Medical Association. He received his preparatory training at Milleston Seminary, East Hampton, Mass. He graduated from Yale in 1874. While at Yale he read a thesis, held the Holmes Scholarship and was a member of Delta Psi. He studied medicine at the Harvard Medical School and received the degree of M.D. in 1877. In 1878 he became assistant to the Superintendent of the Boston City Hospital and the following year House Surgeon. In 1880-81 he was resident physician at Block Island. He made three trips abroad for study during the years 1877 to 1883 devoting his time especially to the study of surgery and pathology at London, Berlin, Vienna and Heidelberg. He was made by examination in 1881 Member and in 1883 Fellow of the Royal College of Surgeons, London.

Dr. Platt began the practice of medicine at Baltimore in 1881. Shortly after he was appointed visiting surgeon to Bay View Hospital and demonstrator of surgery at the University of Maryland. He was appointed in 1886 superintendent and surgeon of the Robert Garrett Hospital for children retaining this position to the time of his death. He gave the best that was in him to building up and directing the medical work of this institution. Its success is a fine monument to his devotion, untiring zeal and ability.

On December 4, 1889, Dr. Platt was married to Mary P. Perine of Baltimore, daughter of the late Glenn Perine. They had four children, Washington, Lucian (who died of influenza at Camp Humphreys in 1918), David P. and Mary (now Mrs. C. Chauncey Hall).

Dr. Platt's death came at the end of a long illness from hypertensive cardio vascular disease. Eight years before his death hypertension was discovered. At that time he suffered from brief attacks of palpitation and precordial discomfort due to the occurence of numerous auricular premature contractions. These unpleasant symptoms were easily controlled by a reasonable modification of his very active life and he was able to attend to his medical duties uninterruptedly until June 1921. He then began to have attacks of very severe angina pectoris and was obliged to spend several months resting quietly at home. During the autumn and winter he was better but his activities were considerably restricted. In August 1922 he had a particularly severe attack of angina followed by a prolonged period of prostration. After a few weeks in bed he recovered from this attack and for a while improved. However, on October 28th, two days before his death there was a sudden aggravation of his symptoms due it was thought to occlusion of one of the branches of the coronary artery.

Dr. Platt was one of the prominent practitioners of Baltimore. He held no academic position and wrote little so that he was not widely known but all of his associates and a wide circle of friends fully appreciated his unusual medical ability and his fine character. When as a young man he came to Baltimore he had a preparation in medicine that few could equal. He was a modest and retiring man who had no taste for public appearance nor for the fascination of the

lecture room and meeting hall. He was satisfied to do his daily work ably and conscientiously and did not reach out for the larger honors that came with successful teaching and writing although he was unusually fitted by his training for such a career. He was a man of engaging personality and high character. He was devoted to his patients and his hospital, never sparing himself when the interest of either was concerned. While unusually modest and diffident about his own attainments he was always solicitous to appreciate merit in others. He was especially fond of young men, interested himself in what they were doing and encouraged them by confident predictions of future success. He spoke well of other doctors, did not envy them their success and was quite free from the petty faults of inuendo and praise with reservations. He was a man of wide culture and a lover of the out-of-doors, therefore a delightful companion. He was kindly and considerate of all. He showed the fine temper of his character throughout his illness. He realized fully the gravity of his symptoms and yet was always cheerful, taking an admirably impersonal attitude towards his illness. He did not revile fate nor pass with bravado as one indifferent to the flings of fortune, but accepted the situation simply and uncomplainingly facing what had to be faced with the calm unostentatious smile of true courage. It was a bit uncanny at times to listen to his shrewd and interested comments upon his own symptoms, stated in the most impersonal way as though he were discussing some interesting patient.

Though in general not addicted to attending medical meetings, Dr. Platt was devoted to the Climatological Association and seldom missed a meeting. Although at the time far from well he attended the 1921 meeting at Washington. He presented a number of interesting papers before the Association, among others, on the climate of St. Maritz, Upper Engadine, Switzerland and on the influence of climate and season on the manifestations of normal and abnormal activity.

JOHN H. PRIOR 1859-1923

John Pryor was born just before the Civil War and his young life was surrounded by an atmosphere of tense feeling and pronounced conviction on all subjects. His natural intellectual endowment was good and reacted to his surroundings.

When he chose medicine as his profession, he put his whole strong personality into his work and was soon recognized as a power in the profession. In addition to his scientific interest in his work there was also always present an altruistic idealism which dominated his work and made of it a blessing to his fellowmen.

He first conceived and then with the cooperation of some of his confreres in the local profession converted the sick ward of the old County Home into the Erie County Hospital which was a very flourishing and well conducted hospital for years. He early showed a great interest in tuberculosis and in the care of tuberculous people and soon developed a tuberculosis ward in the Erie County Hospital which was a model for any city to follow. His work was not confined to this locality, however. He spoke with such vigor and conviction that he soon convinced many in the state that a State Institution was necessary and the State Medical Society under his leadership convinced the State Legislature of this necessity, so that when Raybrook was established he naturally was chosen as its medical superintendent, which position he held for several years. He was too much interested in the practice of medicine to be contented with a position so decidedly administrative in character, so he resigned that position.

His interest in tuberculosis, however, was so great that it was not long before he convinced the profession of Buffalo and then the city fathers that we should have our own tuberculosis sanitarium for the treatment of incipient cases of pulmonary tuberculosis and the Sanitarium at Perrysburg was established. On one of his trips to Europe he visited the Rollier establishment in Switzerland and spent several months there studying his methods. So fully convinced was he of the value of the procedure that he returned to Perrysburg and established the first scientific sunlight treatment in the United States on the principles of Rollier. The value of this method is now

fully established and its widespread adoption is due to the persistence of Dr. Pryor.

Of course, he was one of the prime movers in the establishment of the National Tuberculosis Association and our local Tuberculosis Association and the vigorous Anti-tuberculosis Campaigns that have been carried on from time to time.

Dr. Pryor was elected to membership in the Climatological Association in 1905. He has been constant in attendance at its meetings and when he spoke on any subject under discussion always had something of interest and value to present. He will be missed greatly at its sessions.

In the death of John H. Pryor, Buffalo has lost one its most public spirited citizens who was always interested in whatever was for the benefit of the city at large and especially in those projects which made for the preservation of the health of the individual or provided for the restoration to health of those who were sick, and the medical profession has lost a member who was always one with those who were interested in whatever tended to raise the standard of medical education and of the ethical conduct of the members of the profession.

Dr. Pryor was always a leader—he was never satisfied to be a private in the ranks. Whenever a new proposition was brought before the Medical Societies for action, he gave the subject thought and after he had made up his mind he took a strong stand for or against and used all his eloquence (he was a great orator) on the side he advocated and always had a large and devoted following.

In addition to his deep interest in the general welfare and that of the medical profession in particular, Dr. Pryor's nature was too many sided to be confined to professional and altruistic work and he always manifested much enjoyment of music and literature, especially poetry and drama.

Dr. Delancey Rochester.

PRESIDENT'S ADDRESS

By CHARLES W. RICHARDSON, M.D.,
Washington, D. C.

Members of the Climatological and Clinical Association:—

Our association has within the past few years taken up a dual activity, viz., that of the scientific study and research in the field of climatology; and the later function of stimulating clinical investigation and research.

Our anchorage is so safe, our past history so glorious in great achievements, the results thereof so keenly appreciated that we may safely utilize some of our reserve energy and capitalization of scientific knowledge in that field of endeavor which cannot have too many earnest workers.

We have in this organization, especially among the more recent membership, alert minds, well trained in clinical medicine, who, no doubt, will assist us greatly in developing the clinical aspect of the association's activities.

There has been an important question which ever and anon from my own experience and from observation of others, has passed through my mind during the past few years, and no doubt has been the experience of many of those among my hearers; and that is, are we today carrying out and interpreting the clinical bed-side investigations as we should, and are we teaching them in proper sequence to the aspiring classes that come under our tutorage?

Are we following up and adding to our clinical research work all those advantages accruing from careful persistent clinical detail investigation; or are we placing too much emphasis on mechanical and laboratory methods of diagnosis?

Contemporary medicine has made many of its remarkable advancements through the aid of laboratory investigation. To attempt to practice clinical medicine without its aid would be futile; to attempt bed-side investigation without its aid would be denying to the patient the advantage of modern clinical knowledge. What

we distinctly need as clinicians is to appreciate the fact that the laboratory methods are to be employed as aids to clinical medicine, not as methods to simplify and make easy the perplexing problems of diagnosis. I fear two of the most frequent faults in connection with laboratory findings is the improper interpretation of the findings through the want of knowledge of their clinical significance; and the too ready acceptance of supposed laboratory findings in establishing a diagnosis.

Our immediate predecessors in clinical medicine were men who had been taught and further developed in themselves the art of careful clinical investigation and thoroughness in clinical detail. The circumstances which surrounded them, and the lack of many features which now makes clinical investigation fairly easy, caused them to pay close attention to signs and evidences which the clinician of today is so apt to throw into the discard. If these men of forty years ago, with their superb clinical experience had had our present laboratory facilities, what giants as clinicians they would have been.

We of the present day, no doubt in practice and teaching are accepting, and following too closely the laboratory results, at times I fear, at the expense of our own clinical knowledge. There is no question that the laboratory findings are a most valuable asset in the investigation of many of the obscure cases that come under our observations, but we must always properly evaluate them in relation to our carefully made clinical investigation. We should not permit laboratory findings to overweigh and mentally over-balance our well-worked out clinical findings, but should consider them well, place the proper valuation over all, and wherein they do not harmonize, realize that laboratory results are human and that pathology may alter these results as well as physical signs and evidence.

I should not wish to be considered for one moment less of an adherent to the laboratory than a clinical man, for I am too well aware of the wonderful aid, and, at times the almost complete revolutions in one's clinical view that may be brought about through laboratory results. I also have had personal experience wherein a too frank acceptance of laboratory findings has later taught that a closer and unswerving attachment to the clinical findings would have demonstrated their correctness.

What I wish to plead against is the tendency that I notice here and there, in order to avoid the more tiresome clinical investigation with the knowledge and study it entails to have the way made easy through extensive laboratory investigations.

I fear the tendency which has been developed among some good clinicians of obtaining extensive laboratory findings before they turn their minds to the clinical phase is not only leading them into doubtful clinical practice, but is also furnishing to their juniors, who are not so well developed along the lines of clinical investigation, into bad habits, which in the latter is apt to bear barren fruit.

I cannot conclude these introductory remarks without referring to another important phase of medical research, animal experimentation, which is being jeopardized by the increasing and constant activity of the so-called anti-vivisectionists. It behooves every member of this organization to be constantly on the alert and everlastingly at work at refuting and scientifically combating the assertions of these people. Everyone should be prepared with well established facts and data so as to combat and confuse these people in their generalities, mis-information and mis-statements. It is only through combined efforts, through the duty that everyone feels incumbent upon him, to aid in the cause that will prevent success at any one point by the antis. Education among the laity as to the advantage accruing from animal experimentation to human and animal life is necessary in order to lessen the recruits to the cause of anti-vivisection. As all present so well know, it has been largely through animal experimentation that the present status of modern medicine has been attained. Such being the case why should we hesitate in placing it clearly before those who have not the advantage nor ability to recognize its advantages as we of this favored profession? It is also the duty of the members of this organization, and through you, your county and state medical associations to educate your representatives in Congress so that they will be secure against the false arguments of the anti-vivisectionists.

Gentlemen of the American Climatological and Clinical Association, I wish in closing to express to you my thanks for electing me your president. I now declare the fortieth session open for the transaction of business.

THE USE OF BENZYL-BENZOATE IN THE TREATMENT OF ANGINA PECTORIS.

By ROBERT H. BABCOCK, M.D.,
CHICAGO, ILL.

No attempt will be made in this brief paper to give the literature pertaining to the use and action of this particular benzyl ester further than to say its mode of action, as pointed out by Macht, is to relieve spasm due to contraction of smooth muscle fibre. Hence it has been employed largely in cases of renal colic, bronchial asthma, etc. So far as I know the only article dealing with its use in angina pectoris is a paper by Dr. A. B. Spach of Chicago, which article appeared in the Illinois State Medical Journal in January, 1921. In that paper the author reported six cases in which more or less complete relief followed the administration of the remedy. In one of them the blood pressure was low, 100 systolic, whereas in the others varying degrees of hypertension existed.

My attention to the use of benzyl-benzoate in Heberden's angina was first called by my office associate, Dr. Bayard Holmes, Jr., who stumbled on to its ability to relieve this formidable affection through prescribing it for renal colic. The patient, Mr. T., was a Jew of 53 whom I had treated years earlier for sclerosis of the heart and arteries giving rise to attacks of precordial pain and dyspnea with, as I now recall, rapid and arrhythmic action of the heart. The man was so neurotic and emotional that I could never be quite certain of the exact significance of his attacks of pain. It is likely, however, in the light of subsequent events that his precordial pain was angina pectoris. At all events when after the lapse of several years renal colic developed Dr. Holmes, who saw him in an attack, prescribed benzyl-benzoate with the result that it relieved not only the colic but also his angina. This observation by Dr. Holmes antidated and was wholly independent of the article by Dr. Spach and was never published but was reported at a clinical meeting of the staff of Lakeside Hospital in 1919, I believe. Since that time Dr. Holmes and I

have prescribed the remedy in approximately twenty instances of what seemed to us typical attacks of angina pectoris. I shall not attempt to narrate the particulars of these cases and shall give a brief account of only five in which decided relief of the pain has followed the more or less continuous use of the drug.

Case 1.

Mr. P., a short thick-set Italian of 53, was seen in March, 1921, because of pain beneath the upper sternal region which prevented his walking for more than a few hundred feet. This soon thereafter became so intense that he remained at home and was attended by my associate, Dr. Holmes. The man was kept at rest in bed for several weeks, his diet carefully regulated and vigorous elimination resorted to in the hope of relieving the indigestion complained of and reducing somewhat his high blood pressure which stood at 220 S. and 120 D. The cardiac findings were those of hypertrophy of the left ventricle without murmurs. At length the doctor decided to try the effect of benzyl-benzoate in thirty drop doses in milk four times daily. Under the effect of this remedy the man became able to walk at his ordinary gait without pain. He kept up this treatment for months and ceased medical attention. A few days ago, in the last days of April, 1923, the man reappeared for examination as he had again begun to feel not so well. He admitted having neglected his medicine for the preceding three months. His blood pressure was 215 S. and 100 D. There was an absence of heart murmurs except a systolic bruit in the aortic area on assuming the dorsal decubitus.

Case 2.

Mrs. N., 68 years of age was seen in September, 1921, because of a retrosternal pain of such severity as to compel her to cease walking after having gone a short distance. Family and personal history was unimportant. Her systolic pressure was 150 and the diastolic 80. The heart was somewhat increased in size and there was a soft murmur accompanying the first tone at the apex. From that time to this she has taken benzyl-benzoate practically without omission and declares it has saved her life. Upon rare occasions she has

had her angina when walking in the evening after dinner. Upon coming to the office after such an experience her systolic pressure has sometimes shown striking fluctuation, some of our records reading as high as 180 and once or twice as low as 132. The diastolic has always remained at 80. We have not been able to explain these differences since for the most part her systolic pressure has stood at about 150. I incline to the view that the higher pressures have been at times when she has suffered from her angina.

Case 3.

Mr. G., a Jewish merchant of about sixty years of age, was first seen some five years ago when suffering from dyspnea with attacks of palpitation and pain in the epigastrium associated as well as could be determined with indigestion and smothering attacks. He presented the usual findings of sclerosed arteries, cardiac hypertrophy and a systolic bruit in both mitral and aortic areas. These were believed to indicate definite cardiac and aortic sclerosis. He was seen at varying intervals, usually of months. At length this man moved to Chicago and was seen by me at his home when suffering much from attacks of substernal pain believed to be typical angina. Attacks of this kind became at length so intense and frequent as to necessitate the daily use of nitroglycerin and even of morphin. His suffering grew so alarming that one night his daughter summoned Dr. Holmes in great haste. It was then that the doctor hit upon the happy thought of trying benzyl-benzoate. It is no exaggeration to say that inside of forty-eight hours the pain disappeared not to return during the three months he lived. That was in 1920, I believe.

Case 4.

Mr. G., another Jewish merchant from an Illinois town was seen in December, 1922, because of a pain in the upper middle portion of the chest which had developed not long before and prevented his accustomed walk to and from his store. He was 61 years of age or thereabouts and presented systolic blood pressure of 124 and diastolic 65 with secondary but moderate left ventricle hypertrophy. A murmur was not present. Besides advice regarding his diet and a

periodic cathartic he was told to take thirty drops of benzyl-benzoate in milk four times daily and to be careful not to attempt to hurry up-hill to his home. He has been seen repeatedly in the past five months and only a week ago. He declares he is now able to walk to his place of business without distress provided he does not hurry and considers his improvement due to his medicine. The heart and vascular findings have not changed materially over the orginal examination.

One additional case merits brief mention. A physician aged 54 years who resided in a northern state was seen by Dr. Holmes in May, 1922, during my absence from Chicago. He stated he had suffered for two years from such intense angina pectoris without relief from any remedy that he was considering the abandonment of his practice. His blood pressure was not high, only 130 systolic and 90 diastolic, but he had a mitral systolic murmur of apparently myocardial, not endocardial, origin. He was told to give benzyl-benzoate a trial, and two weeks later reported entire freedom from his angina. He has not been heard from since.

Unfortunately all the cases in which this agent has been given a trial have not experienced so much benefit. A young Italian with a syphilitic aortitis and free aortic regurgitation has received no benefit from benzyl-benzoate. He is obliged to depend on nitro-glycerin, but when seen a week ago after having taken potassium iodid for two weeks he thought he could walk a little better.

We have records of others who reported no benefit at all, while some say they have been somewhat more comfortable. Accordingly it may be said that the administration of this remedy in the treatment of angina is of the nature of an experiment. Therefore, sufferers from this distressing malady are always informed of the uncertainty of benefit. Its prolonged use over many months has not apparently been attended by unpleasant or harmful effects, and only rarely has a patient complained of stomach disturbance. It is always prescribed in milk or cream and generally in thirty drop doses four times daily.

The remedy is not believed capable of correcting the pathology responsible for the distress. It is strictly paliative and not curative. Neither is it believed that it will ward off the fatal catastrophe which

terminates more or less suddenly practically all cases of organic or true angina pectoris.

The varying and uncertain effects of benzyl-benzoate in cases of angina pectoris bring up the question: Why does it relieve the pain in some persons but not in others and what can be its mode of action? The answer to the latter query is found in part in its property of relieving spasm in certain instances through its action on smooth muscle-fibre as in renal colic. But can this action account for the ability of benzyl-benzoate to assuage or even relieve wholly so terrifying and dangerous a malady as Heberden's angina! The explanation must depend, as it seems to me, on the underlying cause of angina.

There is no intention in this paper to discuss the various hypotheses that have been advanced to explain this often truly agonizing and death-dealing pain. I shall mention only two, therefore, sclerosis of the coronary arteries with its consequent effects on the heart muscle so well known to us all, and Sir Clifford Allbutt's view that the cause is found in disease of the aorta and not of the coronaries. In substantiation of his theory he lays particular stress on the nerve-supply to the aorta in especially its suprasygmoid portion. According to Allbutt, Thoma found Pacinian bodies in the adventitia or outer fibrous sheath of the vessel, while Keith and MacKenzie discovered its root or suprasygmoid base to be richly supplied with sensory nerve endings. Furthermore, the sensory nerves and Pacinian bodies are distributed throughout the whole length of the aorta even to its branches within the abdomen, as, for instance, the coeliac plexus. Based on these anatomical facts Allbutt's argument briefly stated is as follows. The aorta is so constructed as to withstand sudden and under some circumstances severe changes in pressure occasioned by each discharge of blood from the ventricle. During violent exercise these alternations in internal pressure may be very great. Yet the elasticity of the vessel is such that in health the tension is passed on along the course of the aorta without giving rise to subjective consciousness of strain. When, however, the aorta, especially its ascending limb has undergone pathological change, acute, subacute or chronic, the state of things is reversed. Ordinary pressure within the vessel is sustained without the production of

symptoms, but when intra-aortic pressure becomes increased beyond a certain limit, as in walking or from emotional excitement, anger for example, vascular tension becomes such that the middle and outer coats of the suprasygmoid portion or indeed of the whole arch may be great enough to occasion their stretching with consequent impingement upon their sensory nerve-endings. This fact is then telegraphed to the sensorium whence response is sent out through the ganglion of the cervical sympathetic to the system of sensory nerves supplying the upper portion of the chest and even into one or both arms through the brachial plexus. Pain generally felt therefore is retrosternal corresponding to the situation of the aortic arch. When during such an attack of angina the pulse becomes altered in rate or rhythm it is due to stimulation of the sympathetic or parasympathetic which send filiments to the cardiac plexuses.

In support of his hypothesis Allbutt points out that post-mortem examination always reveals more or less obvious pathological change in the aortic coats. He does not deny that the coronary arteries, myocardium and even pericardium may display sclerotic, degenerative or fibrous change, but he contends that pathologists are generally so taken up with examination of the heart that they overlook or give but scant attention to the aortic coats particularly the adventitia. If a few patches of sclerosis are seen on the surface of the intima they are content with noting these and do not cut through and carefully examine the middle and outer coats to detect what pathological change they may have undergone. Almost innumerable cases are cited from pathological reports in support of this contention. In further support of his view of angina being aortic and not coronary in origin he points out the frequency of severe angina in acute aortitis whether due to syphilis or to other acute infections, while typical post-sternal angina is a frequent accompaniment of aneurism affecting the ascending aortic or entire arch. The rich supply of sensory nerve-endings to the sinuses of Valsalva explains the excruciating agony attending aneurism of this portion of the vessel.

Without entering into a discussion of the merits of Allbutt's explanation of the mode of production of angina I must confess that it appeals strongly to me as applicable to the cases I observe. Only

yesterday a man of 58 was examined who stated he was unable to walk any considerable distance without very uncomfortable pain in the center of the chest or mid-portion of the sternum. Without giving details of the findings let me say a fleuoroscopic inspection revealed marked dilatation of the entire thoracic aorta with hypertrophy of the left ventricle. Auscultatory findings were practically negative and blood pressure was 142 S. over 75 D. In such a case the conclusion is inevitable that the aortic coats are atheromatous and lessened in elastic recoil. It seems reasonable to me to assume that increased tension of the vessel must by stretching impinge upon the sensory nerve endings and so occasion the uncomfortable symptoms described.

But granting that to be true in the instance just mentioned how may one explain the varying effects of benzyl-benzoate in the cases previously reported in this paper? Even though the remedy does exert its action on smooth muscle fibre it can scarcely be supposed to relax a vessel stiffened and more or less inelastic. Accordingly benzyl-benzoate can scarcely be expected to relieve angina if due to coronary sclerosis. Neither can these vessels when pathological be expected to respond to tension within the vascular system outside of the heart or to any vasomotor disturbance. The same mode of reasoning would probably account for its failure to help the young Italian P. who has pronounced syphilitic aortitis with regurgitation. The hypothesis which appeals to me, therefore, is that in those instances in which the remedy is effectual there is a spasm of some part of the vascular system responsible for abnormal tension of the aortic coats where sensory nerve endings are most abundant, that is, in the suprasygmoid limb of the arch. Hence retrosternal distress. Under the influence of the daily use of benzyl-benzoate vascular spasm is prevented or kept down to such an extent that pressure within the aorta does not become greater than the diseased coats can withstand without subjective consciousness of the strain. If for instance the distal portion of the aorta be comparatively free from atheroma sudden or undue increase of tension at its base can be passed on through relaxation of the comparatively healthy portion of the vessel and painful stretching of the arch does not take place.

How, one may ask, does the likelihood of spasm apply to the

few cases here so briefly reported? In the instance of Mr. T. who suffered from what were regarded as attacks of renal colic it is not unreasonable to assume that vascular spasm was associated through the action of the parasympathetic nervous system so as to occasion a degree of intra-aortic presure which the vessel could not pass on as should occur normally, and angina resulted. Although the actual facts attending his death a few months later are not known to me it may reasonably be assumed that he succumbed to vagal death as in practically all persons who suffer from organic angina.

In the case of Mr. G. whose daily anginal seizures were almost miraculously abolished I incline to the view that in him also there was vascular spasm depending on action of the parasympathetic, that is, a vagotonia, since before typical angina was recognized his dyspnea and arrhythmia were believed due to gastrointestinal disorder, and treatment addressed to the digestive tract had given relief until a time came when angina developed.

As regards Mrs. N. the one fact suggesting possible arterial spasm is the striking variability in our blood pressure records. It varied from 180 systolic to as low as 132 with a constant diastolic reading of 80 mm. She was generally most free from pain and best able to walk when the systolic figure stood at about 150 mm. She did not complain of abdominal symptoms and yet had to exercise care not to overload her stomach or partake too often of meat. It was necessary also for her to resort to a weekly cathartic in order to keep entirely comfortable. Nevertheless she was emphatic in her declaration that benzyl-benzoate had saved her life.

Why the remedy proved so beneficial in the case of the Italian, Mr. P., of 54, who always showed such a marked degree of hypertension but without any other cardiac findings than left ventricle hypertrophy I cannot explain. If symptoms referable to vagus stimulation were present they were shown only by flatulent indigestion. The same may be said of Mr. G. who has been under observation occasionally for the past five months. He has been partially though not wholly relieved. In future attention will be addressed more closely to the possibility of stimulation of the parasympathetic portion of the vegetative nervous system.

As this paper was being finished a man of 45 was seen who had

consulted several physicians because of discomfort in the midline of the chest at about the center of the sternum. This sense of discomfort did not amount to actual pain and yet it was felt when walking and caused him to stop in order to allow the uncomfortable sensation to pass away. The only factor of possible etiological importance was a soft chancre in 1900. Several Wassermann blood tests had been negative however. An x-ray examination by a competent roentgenologist had shown nothing wrong with the aorta and only slight hypertrophy of the left ventricle. Physical examination showed a pulse rate of 80 in perfect rhythm and a blood pressure of 135 S. with 75 D. All heart tones were clear in the upright position and the aortic second was not accented. In the dorsal decubitus a soft systolic murmur could be heard in the aortic area. The man was then told to hop up and down fifty times after which systolic pressure rose at the end of two minutes to 170 and the pulse rate sank to nearly its previous figure. That seemed to show a satisfactory response to the exercise but in a minute or two the man said he felt the distress in the chest, and on his blood pressure being again taken it was found to be 190 S. with 90 D. As the distress subsided his pressure fell a few points but not to 170 as directly after the hopping test. He was then told to lie down and raise himself into a sitting posture six or eight times. This again produced his distress and raised systolic pressure to 192, the diastolic remaining at 90. The percussion and the auscultatory findings, however, were as before exercise. The effect of exercise seemed to warrant the conclusion that the rise of blood pressure raised intra-aortic pressure to a point which stretched its adventitia sufficiently to pinch the sensory nerves and so occasion his distress. On this hypothesis he was given benzyl-benzoate as an experiment. May 17th he appeared again after taking the remedy for a week. His systolic pressure directly after walking to the office was 162, diastolic 75. After exercise, as before, the systolic rose to 178 with diastolic of 90. His substernal distress was less pronounced than a week earlier. After resting in dorsal decubitus for some minutes until the slight distress had passed his systolic pressure fell to 155 and then to 140 and eventually the diastolic to its previous figure of 75. He had had a somewhat more comfortable week. This test seems to me to account for the distress

exercise raising general arterial pressure, and the benzyl-benzoate tending to counteract such a rise. This man's distress is always relieved by nitroglycerin.

The case just described suggests the thought that were the blood pressure taken during the anginal seizure in all instances it might assist greatly in understanding the state of the vascular system directly responsible for the attack. May it not be a transient rise of pressure in cases displaying normal or subnormal sphygmomanometric figures which causes pain after the manner described by Allbutt? If so the benefit occasioned by the daily use of benzyl-benzoate is readily understood, since taken at four hour intervals during the day its effect on the smooth muscle fibres contained in the blood may persist for a sufficient length of time and to a degree sufficient to prevent or lessen painful stretching of the aortic coats.

Summary.

Benzyl-benzoate has been prescribed for twenty or more persons suffering from the retrosternal pain and pressure feelings characteristic of organic angina pectoris. Of those kept under observation six at least have been so distinctly relieved as to justify daily employment of the remedy for months or so long as benefit is experienced. In some of our cases very slight if any amelioration of pain has resulted, notably in a young Italian with syphilitic aortitis with free regurgitation. In instances of hypertension no invariable or sustained reduction of blood pressure has been observed. In no instance have harmful effects been detected. Administered in milk or cream it does not appear to disturb the stomach. As a general rule thirty drops poured directly from the bottle are ordered four times daily. The remedy would appear to prove efficacious in cases in which vascular tension or spasm produces such a degree of intra-aortic pressure as to pinch the sensory nerve endings richly supplied to the aortic coats in accordance with Sir Clifford Allbutt's explanation of organic angina pectoris.

DISCUSSION

Dr. Rochester: I was very much interested in Dr. Babcock's statement in regard to the use of benzyl-benzoate because I have used benzyl succinate in similar conditions of gastric spasm to relieve pain in the pyloric

region and found it very useful in those conditions in which we previously used belladona.

In regard to the size of the heart, I feel that we should examine the patient carefully not only when lying down but also when sitting up. We get more fully the size of the heart by palpitation and inspection than we can by percussion.

Dr. Ringer: Dr. Babcock's very interesting paper brings to my mind a case which I have at present under my care. The patient is fifty-four years of age and has complained of dyspnoea on exertion together with a sense of oppression and of pain in the chest. She is an ex-tuberculous patient but her present condition is quite separate and distinct from the tuberculous process, which has been quiescent for some time.

Renal examination is to all intents and purposes negative save that she has to get up to void two or three times at night, and the specific gravity of the urine is low, below 1.012. Wasserman reaction—negative. All other results of physical examination are negative save the blood pressure, which shows a systolic two hundred and thirty, distolic a hundred and ten. We believed there was probably an anginal condition or else some aortic spasm. No attempt was made to differentiate between the two.

Many things having been tried without success, she was given benzyl-benzoate in twenty minim doses four times a day, and almost at once she had distinct relief. This relief has never been complete but she is far more comfortable since she has been taking the drug. Her blood pressure has fallen until now the systolic ranges from 190 to 200. She does not take the benzyl-benzoate with absolute regularity. According to my instructions she takes it for a while and then stops, beginning again whenever any of the unpleasant symptoms come a little too much to the fore. I think that henceforth I shall instruct her to take the benzyl-benzoate regularly as Dr. Babcock recommends his patient to do, and possibly increase the dose to thirty minims four times daily in accordance with his suggestion.

Dr. Babcock: I would like to add to my report,—most of my patients with characteristic angina have shown by X-Ray examination more or less dilation of the aorta either of the arch or of the entire thoracic aorta. I do not wish to deny that some cases of angina may be due to coronary disease, especially coronary thrombosis, but I do believe that excepting in cases of thrombosis sclerosis of the coronaries alone is responsible for the pain but is associated with atheromatous change of the aorta.

The diagnosis of the condition responsible for a chest pain apparently veritable angina pectoris of the Heberden type is often very difficult. For instance, I saw a man of middle age who sought relief from a pain in the upper left chest so intense as to prevent walking. When told to place his hand over the exact seat of his pain he placed it upon the left anterior pectoralis muscle and then said he had found that he did not have his pain if in walking he carried

his left hand in his side coat pocket. After the extraction of two abscessed teeth he lost his angina which was as previously surmised merely a myositis of the pectoral muscles.

When benzyl-benzoate relieves it is not curative and its administration moreover is purely erperimental. In addition my patients are given orders to be careful regarding exercise and are instructed with respect to habits, diet, etc.

ANGINA PECTORIS

By COLLINS H. JOHNSON, M. D.
GRAND RAPIDS, MICHIGAN.

My purpose in writing this paper is to call attention to the possibility and importance of making a diagnosis in angina pectoris at a time when recovery of the patient is possible, instead of waiting as we formerly did with tuberculosis until an advanced stage was reached when restoration to good health was not to be hoped for; but our former ideas of the etiology and symptomatology must be revised if we wish to diagnose these cases in their incipiency. McKenzie states that there are two broad groups of angina pectoris:

1. Cases associated with disease of the cardio-vascular system and

2. Those in which there are demonstrable cardio-vascular lesions, but which seem to be due to toxic or reflex causes.

Many of the conditions causing angina pectoris are chronic processes which do not give rise to well marked symptoms until they are fairly well advanced, but even when due to organic lesions, many cases can be greatly benefited and life prolonged fifteen or twenty years or more. Occasionally the attacks may wholly disappear and the case end in complete recovery.

It was formerly thought that pyloric stenosis in infants was caused by permanent narrowing of the pylorus, but we now know that many severe cases are due to spasm which may be controlled by atropin with recovery of the patient. In the same way it would seem that in angina pectoris from one cause or another the circulation in the branches of the coronary arteries may be interfered with to such an extent as to prevent the necessary flow of blood through the vessels long before the occurrence of pathological changes in their walls. Autopsies on fatal cases of angina pectoris have shown normal heart muscles and absence of macroscopic disease in the coronary vessels; and severe attacks of cardiac pain may occur in

the absence of any demonstrable lesions in the coronary arteries, aorta or myocardium. Does not the prompt relief frequently given by amyl nitrite or nitroglycerine and the permanent recovery of some of these cases warrant the conclusion that the symptoms are often due to perverted physiological function long before the onset of organic changes in the tissues?

There are no physical signs characteristic of angina pectoris. The diagnosis must be made from the history and symptoms. Such physical signs as are present are indicative of associated conditions. The heart may be normal in size, there may be no arrhythmia, no murmurs, no peripheral sclerosis; and while, as McKenzie has pointed out an attack may be accompanied by a sharp rise in blood pressure, it may be normal or sub-normal during or between attacks. All abnormal physical signs may be wanting and yet the disturbance in the coronary circulation may be severe enough to cause death. Robey, Williams, Longscope, Smith and others agree that even the electro-cardiograph does not always give conclusive evidence although often of great value, and the latter states that in cases of angina pectoris which do not show other signs of cardiac sclerosis, we are unable to obtain any constant help from electro-cardiograms.

In a good many cases, however, in the first group mentioned, angina is associated with other evidence of cardio-vascular disease. Hypertension is not uncommon. Osler says it is the rule, and for some reason the course is often more benign than in the low pressure cases seen in advanced senile arterio-sclerosis.

When cardiac hypertrophy, aortic disease, general arterio-sclerosis or other well marked cardio-vascular lesions are present, we may suspect a case of thoracic pain to be one of angina pectoris. But even in these cases the characteristic pain behind the lower sternum radiating to the neck, shoulders, arms or back with a sense of constriction in the thorax and a feeling of impending death, may be wanting. The pain may be referred to various parts of the abdomen or lumbar region and simulate the pain of acute indigestion, gall stones, renal calculi, pyloric stenosis, etc. The symptoms referred to other organs than the heart may be so severe that the real diagnosis is overlooked for a considerable time. Especially is this the case when the pain is accompanied by digestive disturbances

which may be either an exciting cause or the result of the attack. I have known such cases to be treated by a stomach specialist for a long time without reference to the cardio-vascular lesion.

These cases should be put to bed for a few days of careful observation and given a therapeutic test with nitroglycerine or amyl nitrite. Any pain in the chest or upper abdomen that comes on in paroxysms, that is brought on or aggravated by exercise, and that is relieved by rest and nitroglycerine should be called angina pectoris until proven otherwise. If the patient is over fifty and has any demonstrable cardio-vascular lesions, angina pectoris is all the more probable.

A careful study of the case will show that sooner or later there is more or less pain behind the lower sternum and that muscular effort, strong emotion or indigestion are the more common exciting causes.

There is no such thing as false or pseudo angina. Many so-called cases are true angina and valuable time is lost because of failure to recognize them.

But, it is to the second group, cases of angina pectoris not associated with demonstrable cardio-vascular lesions, that I want especially to call attention and I wish to report three illustrative cases.

Case I.

Twenty years ago I was called to see a man whom I found to have an attack of hemiplegia involving the right arm, leg and face. He was then forty-eight years of age and I naturally concluded he had had a cerebral hemorrhage. To my great astonishment, within twenty-four or forty-eight hours he began to acquire the use of his arm and leg and in three or four days they were about as useful as ever. His speech returned somewhat more slowly, but he resumed work about as well as ever in a week or ten days. We, therefore, concluded that his hemiplegia was caused by spasm of the cerebral vessels due to some reflex or toxic condition which in his case we thought was chronic constipation, as a thorough physical examination disclosed no other condition that we thought could have caused the attack. He remained well for about six months when he had another similar attack which cleared up about as quickly as the first one. A

year later he had a typical attack of angina pectoris. This was followed by more or less sub-sternal and epigastric distress for several months, especially on exertion, so he gave up work for a year. The fact that his distress was frequently evident after a meal, as well as after exertion, and that it sometimes seemed to be in the epigastrium instead of the sub-sternal region, led him to believe that his trouble was due to indigestion, and as his upper teeth had been in poor condition for a long time, he had them extracted so that he could wear a full upper plate. A number of the teeth were found to be abscessed. Almost from that moment he began to improve and all symptoms of angina pectoris soon entirely disappeared. At the end of a month he resumed work and has been perfectly well ever since. He is now 69 years of age with a blood pressure of 125-80 and one of the youngest men of his age in Grand Rapids.

Case II.

About four months ago I was consulted by a man sixty years of age who retired five years ago and had led quite an indolent life since. He was fifty pounds overweight and his only complaint was that for about a year he had had occasional distress in the epigastrium after exercise or a full meal and occasionally a sense of constriction across his chest which always disappeared promptly with rest. An x-ray of his teeth showed that several were abscessed, but he refused to have them out. He had been treated a good deal for indigestion. Examination of his stomach contents, heart, urine, blood pressure, blood, bile, an x-ray of his heart, and on electrocardiogram, were all negative. I had about concluded that his trouble was due to angina pectoris when he had a fairly typical attack. He still thought, however, that his trouble was of gastric origin, so he went to a well known health resort where all sorts of examinations were made with negative results and he was treated for nervous indigestion. His sub-sternal and epigastric distress did not improve and he returned home about two months ago. The next day he went to his office, indulged in more exercise than he had been accustomed to for several months and had four abscessed teeth removed. At nine o'clock that evening I found him having a very severe attack of typical angina. His pulse was rapid and week and

his nails and lips blue. His heart failed rapidly and he died at three o'clock in the morning. An autopsy showed no macroscopic evidence of disease. His heart was no larger than was normal for a man of his weight. The coronary arteries were not sclerosed. The aorta was normal. There is no question in my mind but that he died of angina pectoris induced by the action of toxic material forced into the circulation by the extraction of abscessed teeth upon a heart muscle which had already been greatly weakened by chronic toxemia, indolent habits and overweight. If the autopsy showed that his coronary arteries were diseased and the blood supply of the heart thereby impaired, there would be no question as to what caused his death; but cannot the same condition of insufficient blood supply be caused by constriction of the blood vessels of the myocardium due to toxic or reflex causes?

Case III.

F. K., aged 65. For a number of years has been troubled off and on with indigestion for which he has consulted a good many physicians. It was frequently attended by more or less distress in the epigastric region and he was put upon a rigid diet which reduced his weight in the course of six months from 175 to 140 pounds. But his strength and nutrition were considerably impaired thereby and his distress not at all relieved. On arising hastily from bed one morning, he was taken with a severe attack of pain and I found him suffering from a typical attack of angina pectoris. On close questioning he admitted that the pain which he had always called epigastric was really under the lower part of his sternum whence it occasionally radiated to the right or left arm and sometimes to the back. He was relieved to quite an extent by amyl nitrite and large doses of nitroglycerine, but required one hypodermic of morphia and atropia. The acute pain was followed for several days by a dull ache which gradually disappeared, but he remained in bed for two weeks, as he found that effort caused a reappearance of pain. He was unable to take much exercise for a couple of months and did not resume his usual work until two or three months later. As soon as it became safe, he had his tonsils and four abscessed teeth removed. In addition he has been careful to have one or two good bowel evacuations

a day. That was a year ago. Since then he has attended to his usual duties and has had no further distress of any kind. An x-ray shows that he has a small heart and I believe that his first attack of severe angina was due to the impaired nutrition of his myocardium brought on by rigid dieting, combined with more or less toxemia from chronic constipation, abscessed teeth and diseased tonsils.

McKenzie says that when a healthy person runs until his heart becomes exhausted, he is compelled to rest because of a sensation of oppression or pain in his chest and that the pain of angina pectoris may be looked upon as an expression of exhaustion of the myocardium. This may be due to anything which causes an inadequate supply of blood to it, such as overwork, worry, poor food, infections of various kinds, intestinal stais, etc. as well as valvular defects or other organic cardio-vascular diseases.

Conclusions.

I. There is a stage in angina pectoris where the only change is in the vessels of the coronary tree with no lesion sufficiently definite to cause physical signs or electro-cardiographic changes. These vascular changes may produce physiological disturbances sufficiently severe to cause death.

II. The physiology of the heart may be so profoundly affected by an accumulation of metabolic products in the heart muscle, due to temporary or permanent derangement of the coronary circulation, that the pain of angina pectoris may result. This may occur in hearts in which there are no structural lesions as well as in those affected by disease.

III. While the majority of cases of pronounced angina are due to organic conditions such as diseases of the coronary arteries, aorta, mitral stenosis, myocardial degeneration, hypertension, etc., attacks may also be caused by functional disturbances of the coronary circulation due to toxic or reflex causes.

IV. Angina pectoris is a cardiac pain due to coronary spasm or constriction

V. The disease in many cases is curable especially if the exciting cause can be removed and the circulation in the coronary arteries be improved by digitalis and other measures before they become

seriously diseased. The therapeutic test with nitroglycerine at the time of an attack is useful in differentiating true angina from other conditions. The distinguishing feature is the fact that the pain is brought on or increased by exertion.

VI. There is no such thing as false angina. It is either angina pectoris or it is not angina pectoris. Call any paroxysmal pain in the chest brought on by exercise angina until you prove it to be otherwise.

DISCUSSION

DR. ROCHESTER: I was much interested in this paper. I don't believe the three cases referred to were any of them angina. Pain in all of these cases was in the stomach. I feel that a great deal of the pain connected with cardiac disease due to gastric indigestion can be relieved very promptly by carefully selected diet and the use of insoluble alkali.

DR. DUNHAM: I would like to emphasize the fact that the classification of angina should be based upon etiology. I feel that our efforts should be applied to qualify the various complaints which are used under the term of angina. If we will do that we will come closer to fully understanding each other. This paper brought out one very important point and that is that we may have all the symptoms of angina from a focal infection. This is really a phase of focal infection which I have been overlooking. I am very much interested in Dr. Wale's remarks. The work of Dr. Webb where he emphasizes the importance of the nasal sinuses as a possible focus of infection is of the greatest significance. It is one of the biggest practical points that I have learned in my chest work recently.

DR. ROBERT WILSON, JR.: Recently I have had occasion to analyze the records of one of my patients through a period of several years for the purpose of correlating, if possible, the symptoms presented at various times with the blood pressure readings.

This patient has a dilated aorta and hypertrophied heart, and he has been subject to attacks of precordial pain which at times radiates into the left arm. His blood pressure has varied through quite a wide range, and somewhat to my surprise I found that his anginose attacks occurred just as frequently when his blood pressure was low as when it was high.

The theory that angina is due to coronary spasms seems hardly satisfactory. That spasmodic constriction of a thickened coronary artery may occur is purely hypothetical and certainly would appear to be very improbable. This conception is advanced also in explanation of such other transitory vascular phenomona as aphasia, but in this case it seems more reasonable to assume that we have to deal with a narrowing of the capillary vessels, or possibly of the venules, with a resulting congestion of the area involved and consequent

aedema. May we not in some cases of angina, at any rate, find a similar explanation?

Dr. Johnston: When I became interested in hearts, I read Dr. Babcock's book and never will question anything that he wrote in that book, but I am in good company when I attribute the ethiology of angina pectoria to coronary disease. I think that angina pectoria has certain typical symptoms, under the lower part of the sternum. Those are the attacks we ought to call angina. We should not call a pain of indigestion angina. Angina pectoria can cause coronary disease. A few days in bed will differentiate indigestion. We use the term angina as a basket where we throw chest pains. we should limit the word angina.

SILICOSIS IN ONTARIO GOLD MINERS.

By JABEZ H. ELLIOTT, M.D.,

TORONTO, CANADA.

The studies of miners' phthisis in the gold miners working in the silicious deposits in South Africa and Australia, the reports of Lanza and Childs on a large number of cases found in the silicious zinc miners of Missouri, and the studies of Jarvis, Baldwin, and others in the granite industry of Barre, Vermont, have demonstrated the prevalence of silicosis in men working for long periods of time in quartz, granite and other silicious rock.

Extensive mining operations have been carried on for several years in Northern Ontario in the silicious gold bearing ores, and the industry is developing and extending rapidly. The gold bearing ore shows about twelve per cent. of quartz. Most of the mining is done in schist, which is highly silicious, but as it does not show the same sharp fragmentation as quartz, it has been presumed that its fine dust does not cause as great irritation or damage in the lung when inhaled. However, the report on the dust samples show the particles have uniformly jagged and sharp edges and are occasionally needle-like in form.

Pathology.

The recent work of Mavrogordato, who has been studying experimental silicosis in connection with an investigation of the disease in South African miners, would seem to lead to the conclusion that the silicious dusts are non-harmful from the mechanical point of view and that previous theories as to the ætiological significance of sharp edges and the acicular shape of dust particles have now been discarded. It would appear from his work and that of other investigators that small particles of silicious matter are taken up by the tissues in the form of collodial silica and confer upon the cells which ingest them the property of protection from autolysis and digestion in the lymph. It is suggested that such particles are slightly soluble

in the alkaline tissue juices and that alkaline silicate so formed pervades the cell, preserving it as water glass preserves eggs. The cells containing silica particles flock together and form small masses, which may be called pseudo-tubercles and the similarity of silicosis with early stages of tubercle is remarkable. These pseudo-tubercles later undergo fibrosis and cause lymphatic obstruction.

The suggestion of the Director of the Division of Industrial Hygiene, Provincial Board of Health that it seemed desirable to know whether mining operations in Ontario were causing miners' phthisis in the underground workers was kindly received by both the Provincial Health Department and the Department of Mines, and the Division of Industrial Hygiene was requested by the Chief Inspector of Mines of the Province of Ontario to undertake such an investigation.

The x-ray plates have been read by Dr. G. E. Richards of the Toronto General Hospital and, through the courtesy of Dr. R. R. Sayers, Chief Surgeon, U. S. Bureau of Mines, by Dr. Henry K. Pancoast, Consulting Physiologist, U. S. Bureau of Mines.

Two separate studies were made:

1. As to the presence of silicosis.
2. Dust counts at the breathing level of the men engaged in various operations.

1. The Presence of Silicosis.

Silicosis is a disease of slow development. Few cases have been found in those under five years at work. The Barre investigations showed that of 427 cases of silicosis studied there were six first stage cases under five years and three second stage cases.

A large proportion of the miners in the Ontario camps had been engaged in mining elsewhere. These were eliminated from the present study.

Operations above ground do not expose the men to air heavily dust-laden. Men engaged in such work were not studied.

These considerations led to the adoption of three requirements:

(1) Had worked in the Porcupine camps over five years.
(2) Had not mined in any other camp than Porcupine.

(3) Had worked underground during all or the greater part of that time.

Of some 1,800 men employed by one mining company the mine surgeon was able to find only eleven men who met these requirements. Careful history taking, clinical examinations and study of stereoscopic roentgenograms (repeated in some cases) led to the following *conclusions:*

Four showed definite evidence of pulmonary silicosis, and in three of these the stereograms showed mottling in the apices which might possibly be considered as tuberculous deposits.

Two showed an early stage of silicosis (peribronchial fibrosis without lung involvement).

Three showed moderate peribronchial fibrosis, which may or may not have been due to dust.

Two were considered free from silicosis.

Of the four with well-marked silicosis, three are machine drillers and the fourth has been lumberman and shift boss underground for eight years.

Of the two with early silicosis, one has been machine driller five years and mucker four years; the other, mucker four years and machine helper one year.

Of the three with peribronchial fibrosis, possibly a dust fibrosis, one has been scaler one year, timberman three and one-half years, with mining experience elsewhere. One has driven electric motor for seven years underground. One has been mucker, driller, helper and cageman.

Of the two with no silicosis, one underground six years, has been mucker four months, driller four years, machine helper ten months; one has been timberman ten years.

Case Histories.

No. 182—Italian, age 31.

Occupational history: Farming five years in Italy, at fifteen came to Canada; railroad construction three years, back to Italy for two years, returned to Canada 1912, ten years in Porcupine camp, surface four months, hammer drills two years, water drill six years, shooting blast two years, foreman of muckers.

Personal history: No evidence of contact with tuberculosis.

Previous illness: none before mining; since mining, "strained" right chest wall, necessitating lost time from work of four or five days; LaGrippe fifteen days; occasional shooting pains in the chest; frequent colds in the winter time during the last four or five years.

Present condition: Symptoms: Hard, resistant cough on going to bed and in the morning until about nine o'clock; little in the day time; considerable expectoration both at night and in the morning, generally clear but sometimes black, always worse when he has a cold; has dyspnœa on slight exertion, walks upstairs slowly, says he cannot work as well as he did, has felt weakened for past two years, never fainted; put on weight following 1914 but since 1919 has lost four or five pounds; has pain in chest when he gets a cold, at times without a cold has pain "like pins" in anterior chest, sometimes hurts to breath, the chest feels tight when he has a cold; some days feels very well but if it rains has pain across the back and right shoulder for three or four days, with changing weather has pains in the calves of legs and ankles; no night sweats.

Physical examination: No cough, no pain; dyspnœa marked on exertion; says "can't run very far for last two years." Well nourished; subcutaneous fat plus; large, full chest; subcostal angle 90°; no depressions; antero posterior diameter increased; expansion 36½-38 inches; chest fixed type; superficial cardiac dullness small area near sternum left side, fourth rib, fifth interspace; apex normal; lung borders below move freely; apices full width; lung fully resonant throughout; vocal fremitus, no change; vocal resonance, no change; respiratory murmur normal throughout; few marginal crackles right axilla.

Blood pressure, systolic 122, diastolic 94.

Pulse rate, 76.

Heart sounds, normal.

Tonsils, enlarged but smooth.

Sputum examination: Tuberculosis, negative; dust, positive.

Radiology (Dr. G. E. Richards): The heart and arch of the aorta are normal in size, shape, and position. The diaphragms are smooth and even, and movement is not restricted. The hylus shadows are rather heavier than is considered normal, and many

calcerous glands and nodes are present in each. Radiating out from the right side, the bronchial tree is distinctly heavier than normal, and the whole lung tissue on this side is studded with a number of minute calcerous nodules, which are fairly evenly scattered throughout the entire periphery on the right side. On the left side a similar appearance is present, and the result is to give the lung the appearance of being studded with fine discrete densities resmbling sago grain. There are no localized lesions, and no evidence of infiltration in the parenchyma at any point.

Summary: In my opinion, the examination points to the presence of a moderate degree of peribronchial fibrosis, with a miliary deposit of calcareous material throughout the chest. It does not suggest a tuberculous process, but rather a non-tuberculous density, such as is present in silicosis.

Radiology (Dr. Pancoast)—Plate No. 1. Early second stage pneumoconiosis; Hilum shadows increased; Trunk shadows thickened generally; Diffuse mottling throughout most of the right lung and in left lower lobe.

Diagnosis: Extensive bilateral silicosis.

No. 216—French Canadian, age 35.

Occupational history: (1) Previous to mining—bush work at thirteen years of age; at fifteen years, axe work, driving logs until twenty years; prospecting for three years; mining and prospecting mixed for three years until went to Porcupine.

(2) Mining history—(a) Elsewhere—very little during three year period previous; all in Northern Ontario.

(b) Porcupine camp—here ten years; two years prospecting; half this time in mine; timbering four years; six years shift boss underground.

Family history: No tuberculosis in family.

Personal history: Slept in next bed to a case of tuberculosis while in camp six years ago.

Previous illness: No sickness before began mining; since mining has had typhoid in 1910 and rheumatism in 1920, when he was in bed for two months; suffered from some shortness of breath after that; has colds four or five times in the winter, none in summer time.

PLATE I.

Case No. 182
Plate No. 99593
Extensive bilateral silicosis.
Early second stage pneumoconiosis. (Dr. Pancoast).

Present condition: Symptoms: Coughs only when he has cold in the winter time, with thick, brownish-yellow expectoration; no dyspnœa now; lost twelve pounds when he had rheumatism in 1920 which he has never regained; no pain or tightness in the chest;

when he had a bad cold two months ago had night sweats sufficient to completely saturate night clothes, no night sweats now; continual constipation.

Physical examination: Antero posterior diameter of the chest, plus, with sternum rather prominent; subcostal angle is narrow; chest is rigid; scarcely appears to move on deep inspiration yet the measurements are 32½ to 34½. Moderate general hyperresonance with precordial dullness lost; clavicles prominent—deep fossæ above and below; lower lung borders low and move freely; breath sounds generally diminished—no adventitious sounds; clinically:—emphysema, moderate grade with suggestion of fibrosis.

Sputum examination: Tuberculosis, negative; dust, positive, pus cells, plus plus.

Radiology (Dr. G. E. Richards): The heart and arch of the aorta are normal in size, shape, and position. The diaphragms are smooth and even and movement is not restricted. The hylus shadows are moderately heavy on both sides, and calcereous glands are present in both. In the left several of these appear to have been caseous. Extending out from the right hylus there is well-marked coarse mottling distributed over the entire lung tissue from base to apex. These areas of mottling show further localized areas of increased density, with fibrosis, particularly present in the apex, first and second interspace trunks and extending along towards the hylus shadows in both of these regions. On the left side a similar appearance is present, also distributed over the entire lung tissue from base to apex, with localized areas in the apex, and first and second interspace trunk towards the periphery. The mottling is very coarse, and very widely distributed throughout the entire lung tissue on both sides of the chest. Evidences of slight compensatory emphysema are present at both bases. I believe these are evidences of extensive tuberculosis involvement, but also it is exceedingly probable that many of the areas of mottling which are seen in these plates are due to deposit in the lung, which should be interpreted as anthracosis. These are in the nature of multitudes of small, discrete densities, which are too large to be miliary tubercle, and are too discrete to all be tuberculous areas.

A second set of plates made four months later were read as follows:

The diaphragms are smooth and even, and movement is not restricted. The hylus shadows are both increased in density, the left being much heavier than the right. Calcareous glands are present in both, but particularly in the left. Radiating out from the right hylus, the bronchial tree expands immediately into a lung which is filled with areas of mottling extending from the extreme apex to the extreme base. These areas are heavier in the region of the apex and first and second interspaces than elsewhere, and in this region of the lung there is a suggestion of grouping in clusters, or small conglomerate shadows suggesting localized areas of infiltration. Towards the base the appearance is that of a multitude of fine discrete nodules.

On the left side an almost exactly similar appearance is present, the density being slightly less heavy and less numerous. In the base the lung appears almost clear.

Summary: From a radiographic standpoint, the appearance is that of a wide-spread fibrosis with numerous areas of localized density, which are more particularly present in the upper two-thirds of each lung. In these areas many of the patches which are seen present the appearance of conglomerate shadows such as are formed by tuberculous infiltration in the parenchyma but the distribution is much more extensive, and is bilaterally symmetrical. From the plates alone without any other information, I would not feel justified in a definite differentiation between a tuberculous infection and anthracosis. Knowing the history in this case, I believe the changes are due to silicosis and not to a tuberculosis process.

Radiology (Dr. Pancoast)—Plate II. Second stage pneumoconiosis; dense hilum shadows not increased in extent; trunk shadows rather prominent; right lung: slight mottling in lower lobe; marked diffuse mottling entire upper lobe with considerable density; distribution strongly suggestive of a chronic tuberculous process in addition to pneumoconiosis; left lung: mottling throughout and distribution more concentrated around the hilum suggests pneumoconiosis mainly; apex uncertain for tuberculosis.

Diagnosis: Wide-spread bilateral silicosis.

PLATE II.

Case No. 216
Plate No. 99590
Wide-spread bilateral silicosis.
Second stage pneumoconiosis. (Dr. Pancoast).

No. 2164—Italian, age 32.

Occupational history: (1) Previous to mining—Canada 1906; railroad construction four years; T. & N. O. two years.

(2) *Mining history*—(a) Elsewhere—nine. (b) Porcupine

camp—Sept., 1912; surface one year; mucker one year; running hoist six months; machine helper one year; machine driller two years; contract (in mine) one year; machine drills at present, wet.

Family history: No tuberculosis in family; father died after rain and cold, sick three days; caught pneumonia (?).

Personal history: Was in lumber and railway camps for six years but does not know of any special contact with tuberculosis.

Previous illness: Never sick before he began mining; since mining has lost only one day from sickness.

Present condition: Symptoms: Frequently has slight, dry cough which occasionally wakens him at night; worse when he goes from a low temperature to a higher one; small amount of dark expectoration a few times a day; no dyspnœa at ordinary work; says he can run a little but not too much; no loss of weight; when he gets cold has pain in the right shoulder and at times in the sternum; for the past two years has frequently had a "stitch" under the costal margin in front, first on one side and then on the other, has not felt this recently but previously was very marked; no night sweats.

Physical examination: Nourishment, fairly good; subcutaneous fat, slight; color, high.

Dyspnœa, none; "I can run all I want."

Chest, good general contour with angle of Louis and second costal cartilages prominent; clavicles prominent with depressions above and below; subcostal angle 70°; chest expansion, semi-rigid type, good movement below, expansion 34¼ inches to 35½ inches; percussion, good movement lower lung borders; apices, narrow; poor resonance outer third of clavicles; no precordial dullness (superficial).

Pulse, 64; heart sounds, normal; P. M I. fifth space beneath nipple; B. P. systolic, 108; diastolic, 74. Right, 1-3 ribs anterior, inspiration harsh; expiration, prolonged. Vocal fremitus, equal throughout chest. Vocal resonance plus, right 1-3 ribs anterior. Moderate general hyperresonance. Breath sounds, well heard over whole chest posteriorly; few definite sticky rales just above base of spine, right scapula. No tonsillar enlargement.

Sputum examination: Tuberculosis, negative; dust, positive; epithelial cells positive.

Radiology (Dr. G. E. Richards): The heart and arch of the aorta are normal in size, shape and position. The left diaphragm is smooth and even, and movement is not restricted. The right diaphragm is slightly angulated in the region of the dome, indicating failure of expansion of the right lower lobe. The hylus shadows are excessively heavy on both sides. Extending out from the right, the bronchial tree over the entire right side is heavily thickened, and marked mottling is present over the whole of this side. On the left side there is a similar degree of mottling which extends from the apex to the extreme base, and is heaviest in the mid-lung area. This is associated with considerable fibrosis throughout the lung tissue, and on the two sides the appearance is characterized by the presence of large numbers of small discrete densities resembling sago grains. The distribution is even, and presents all the radiographic characteristics of rather well-marked anthracosis with a number of localized areas of increased density, which suggest a localized tuberculous area. These are present in the right upper at the level of the first interspace, in the right mid-lung area, and most markedly of all in the left mid-lung area at the level of the second and third interspace trunks.

The appearance suggests that of a disseminated miliary type of tuberculous infection, and this must be differentiated from anthracosis. The shadows are rather coarse for a miliary tuberculosis and I believe the appearance is anthracosis with other areas which suggest tuberculous infection in the position described.

A second pair of plates made four months later were read:

The heart and arch of the aorta is normal in size, shape, and position. The diaphragms are smooth and even and movement is not restricted. The hylus shadows are both heavier than is considered normal, and both contain celcereous glands and nodes. The interlobar pleura between the right middle and lower is distinctly seen in its entirety. Extending up from the right hylus, the vertical bronchus is much thickened, and expands towards the apex into a definite density occupying the apex and first interspace trunk in front, and extending down to the third rib posteriorly. Throughout the remainder of this side the entire lung is scattered with a multitude

of fine discrete fibrotic nodules resembling in size and shape sago grains scattered over the whole lung field.

On the left side there is a slightly heavier shadow in the apex and first interspace trunk, but apart from this, the whole lung on this side is also covered with a similar fine mottling, which extends from the extreme apex to the extreme base.

Summary: From a radiographic standpoint, the appearance is that of a miliary deposit of either calcareous material or dust particles. The localized shadow in the right upper is that of an area of infiltration in the lung, and this is also true in the left apex to a less extent. In the right apex there is a suggestion of pleural thickening and restriction over the apical cap.

Radiology (Dr. Pancoast)—Plate III: Typical second stage pneumoconiosis. Hilum shadow on the right side considerably increased in extent; left side hidden by heart and arch of aorta, probably almost similarly increased; diffuse fine mottling throughout both lungs, more noticeable on the right with more spots and extending from the diaphragm to each extreme apex; somewhat fewer spots on the left side but distribution just as wide; left apex entirely clear of any evidence of tuberculosis; right extreme apex slightly suspicious; diaphragm on each side shows a slight humping at the inner and middle thirds usually found in this stage.

Diagnosis: Silicosis of both lungs.

No. 2005—Finlander, age 42.

Occupational history: (1) Previous to mining—on farm, nine to eighteen years of age; at eighteen years came to Michigan, iron mines.

(2) Mining history: (a) Elsewhere—travelled extensively; two years iron mine in Michigan; has worked in coal, lead, zinc, gold, silver, and copper mines. (b) Porcupine camp—nine years, with exception of two visits to Finland; has always worked on drilling machine.

Family history: No tuberculosis in family—mother and father both alive and well; brothers, one alive and well, two dead—one flu; sisters, two alive and well, one in Canada.

Personal history: No special known contact with tuberculosis.

PLATE III.

Case No. 2164
Plate No. 99591
Silicosis of both lungs.
Typical second stage pneumoconiosis (Dr. Pancoast).

Previous illness: No sickness before he began mining; since mining he had rheumatism twelve years ago, four months in bed, very severe; since then has had slight pains in joints but never stops work; has frequent colds both in winter and in summer.

Present condition: Symptoms: His cough is loose; sometimes worse in the morning but continues throughout the day; during the past ten years has been getting worse; considerable amount of thick, black expectoration; has had shortness of breath for four years, even when he works or walks, when he must rest for a short time about every twenty minutes, cannot run; in the past eleven months has lost sixteen pounds in weight; no pain or tightness in the chest; occasional night sweats, clothes wet in the morning, with alternate sensations of heat and cold; teeth are gone; cannot eat meat; has no appetite and in the morning feels shaky and dizzy.

Physical examination: Chest rigid; rather flat; subcostal angle 140°; moderate general hyperresonance; lung borders low and do not move well; breath sounds generally diminished, a few sticky rales interscapular right and left throughout inspiration; dullness over hilus to the left of sternum; heart: P. M. I. fifth space, not out; visible pulsation in the neck over carotids and in antecubital fossal; slightly collapsing quality in pulse; blood pressure: systolic, 126-136, diastolic, 68-70; soft diastolic murmur to the left of sternum at fourth and fifth ribs; Wassermann negative.

Sputum examination: Tuberculosis, negative; dust, positive; epithelial cells, positive.

Radiology, Jan. 27, 1922 (Dr. G. E. Richards): Two single plates of the chest received, both of which show very extensive areas of mottling and fibrosis, within almost the entire extent of both lungs. The only part of the lung tissue, which does not show involvement, is the extreme bases on both sides. These areas of mottling present all the radiographic characteristics of old fibrosis, with areas of infiltration involving practically the entirely lung tissue on both sides, with the exception of the extreme base.

This fibrosis is of the type which is consistent with a history of silicosis and which probably is correctly interpreted as representing this condition, rather than a tuberculous process. The amount of deposit is extensive, and is very well marked, but the plates are not stereoscopic and cannot be reported upon in more detail from that aspect.

A second set of plates of the same man have since been received, October 31st, and these confirm the previous report, showing an

exceedingly well marked fibrosis with mottling distributed over the entire lung tissue on both sides.

I believe we are justified in concluding that this is a true anthracosis.

March 10, 1922. A later set of plates give this reading:

Stereoscopic plates were made of the chest. The heart and arch of the aorta are normal in size, shape, and position. The left diaphragm is smooth and even, and movement is not restricted. The right diaphragm shows evidence of adhesions between the visceral layer of the lung and the upper surface of the diaphragm with incomplete expansion in the right base of the lung. The hylus shadow is excessively heavy on both sides. Radiating out from the right hylus, the bronchial tree shows very marked fibrosis and peribronchial thickening with masses of infiltration and mottling over the right apex, particularly in the posterior part of the lung, and a similar degree of fibrosis extends over the whole right side down to the extreme base. On the left side the upper third of the lung is occupied by marked fibrosis with areas of mottling, with localized density throughout this whole region of the lung. The lower half of the lung shows a moderate amount of peribronchial thickening and fibrosis, but is comparatively clear as compared with the remainder of the chest.

Summary: From a radiographic standpoint there is evidence of very marked fibrosis throughout the entire right side and the upper half of the left side. Numerous areas of localized density are present which I believe to be areas of infiltration. I would find it impossible to differentiate the fibrosis and areas of mottling here seen from those produced by tuberculous disease, and believe that a tuberculous ætiology should be considered unless it can be definitely eliminated by clinical methods.

Radiology (Dr. Pancoast):—Plate IV: Advanced second stage pneumoconiosis; hilum shadows considerably increased in density and extent; linear markings and trunk shadows considerably accentuated especially on the right side; diffuse mottling throughout both lungs; an increased amount of mottling in each apex makes the case somewhat suspicious of a chronic tuberculous process in addition; there is considerable general fibrosis throughout both lungs and

probably some emphysema at the bases; both diaphragms show multiple elevations due to the pull of fibrous bands.

Diagnosis: Extensive bilateral fibrosis due to silicosis in the late second or early third stage. (This man had extensive underground mining experience before arriving in Porcupine camp).

PLATE IV.

Case No. 2005
Plate No. 99592
Extensive bilateral fibrosis due to silicosis in the late second or early trird stage. Advanced second stage pneumoconiosis. (Dr. Pancoast).

2. Studies of Mine Dust.

The collection and estimation of dust, the temperature and humidity readings, were taken by Mr. W. E. Greene, chemist in the Provincial Board of Health laboratories, under the supervision of Mr. H. M. Lancaster, the Director.

The three types of apparatus in use for the collection of dust samples are:

(1) The Koniscope—where air is forced against a glycerine plate which catches the dust. This was mainly used in the South African investigation.

(2) The sugar tube in use by the United States Bureau of Mines.

(3) The Palmer Spray apparatus.

The last apparatus was adopted as standard for dust collection by the American Public Health Association. It has many disadvantages, but its consistent use at least allows us to make comparisons between our own findings.

In this investigation all particles in size from $\frac{1}{2}$ to 12 microns were counted.

South African investigations indicate as the allowable maximum, the presence of 300 particles of silicious rock in size from 1 to 12 microns per 1 c.c. of air. This is equivalent to 7,680,000 such particles per cubic foot of air.

Study of the dust counts among other things shows:

(1) That the dry drill causes a higher air content of dust of a size considered to produce miners' phthisis than a wet drill.

(2) The air of upcasts shows a low injurious dust count, due to the high humidity of mine air and the ventilation.

(3) When collaring dry, dust counts are consistently high.

(4) The dust counts in dead ends of drifts and in raises are moderately high.

(5) The amount of harmful dust in the air is similar in all the large silicious rock mines.

COMPARISON OF DUST COUNTS AND WEIGHTS IN THREE PORCUPINE GOLD MINES

No.	Operation	Mine A		Mine B		Mine C	
		Count	Weight	Count	Weight	Count	Weight
1	Upcast air	230,000	.126	227,000	.107	226,000	.118
2	Main travel ways	....	....	227,000	.129	234,000	.106
3	Leyner in Stope	1,673,000	.620	2,881,000	.432	1,506,000	.324
4	Leyner in Drift	2,070,000	.466	2,389,000	.466	1,499,000	.430
5	Wet Stoper in Raise	17,400,000	1.990	12,550,000	1.608	2,550,000	.543
6	Dry Stoper in Raise	221,000,000	15.9	86,000,000	13.00	....	....
7	Mucking in Drift	1,096,000	.276	756,000	.225	824,000	.223
8	Mucking (Taking down back)	916,000	.412	....	....	1,703,000	.229
9	Plugger in Stope	....	....	208,000,000	14.3	....	....
10	Leyner in Raise	....	....	....	....	1,982,000	.284
11	Wet Stoper in Stope	1,704,000	.610	....	....	....	....

NOTE: In some cases of drilling there is considerable variance in the results obtained at the different mines. The ratio of number of particles to weight of particles also varies. This is not surprising, however, when the extent of variable factors met with in dust collection is considered. For example, much depends upon (a) the kind of rock being drilled, (b) the number and type of drills being used, (c) the extent of ventilation, (d) the degree of confinement, (e) the distance the sampling machine is from drilling face, (f) whether sample is taken at breathing level or lower, (g) some men work much harder than others. Samples were taken at breathing level whenever possible and other conditions were kept the same as possible. Any great variance in particular instances will be explained below.

No. 3—Count at B higher due to a plugger operating in some stope.

No. 5—A count higher due to two stopers operating. There was only one in use at Mine B. Mine C result should not be included here as the raise was not yet formed and the Leyner was regularly used. Test taken for comparison with Leyner results under similar conditions.

No. 6—No real dry stopping was obtained at B or C. Samples listed as such at B were dry collaring.

Nos. 6, 10, and 11 are not regular operations and were taken for comparisons only.

Chemical and Microscopical Report on Mine Dust.

Samples of rock from three mines were submitted to the Provincial Assayer, Bureau of Mines, Mr. W. H. McNeill, who reported as follows:

	B	C	D
Silica	49.12 per cent	50.34 per cent	52.04 per cent
Ferrous oxide	5.15 "	5.02 "	5.02 "
Ferric oxide	Trace	0.85 "	1.63 "
Alumina	7.82 "	8.98 "	8.65 "
Lime	8.58 "	7.74 "	7.34 "
Magnesia	4.39 "	4.05 "	4.13 "
Sulphide of iron (pyrite)	7.16 "	5.90 "	4.66 "
Soda	1.08 "	1.16 "	1.24 "
Potash	1.78 "	1.92 "	1.84 "
Carbon dioxide	13.40 "	13.04 "	12.44 "
Water (combined)	1.45 "	1.35 "	1.44 "
	99.93 per cent	100.35 per cent	100.43 per cent

W. M. McNeill,
Provincial Assayer.

The analysis of the dust samples collected from the air near machine drills at work gave this result:

Silica	56.06 per cent.
Iron and Alumina	15.96 per cent.
Lime	6.6 per cent.
Magnesia	1.56 per cent.

N.B. Iron estimated as ferric oxide.

Dust samples "A," "B," "C," and "D" were mounted between glass with Canada balsm and examined under a microscope. Practically all the particles are irregular in outline, having jagged and sharp edges and occasionally being needle-like in form. The smallest particles could not be identified, but they are probably similar to the large pieces. The larger fragments consist of quartz, feldspar, carbonate, silicate leucoxene and iron oxide, etc. They are .1, .2, and .3 mm. in diameter. According to Mr. Ellis Thomson, the numerous smaller particles are .0008 mm. in diameter.

Conclusions and Comment.

The following suggestions were made:

1. That men who have worked five years in mines elsewhere be examined with the fluoroscope if engaged for underground work, and that a yearly examination be made thereafter.

2. That all employes be examined with the fluoroscope at end of five years' underground work and yearly thereafter.

3. That when the fluoroscope reveals possible dust deposit and fibrosis, that stereographic radiograms be studied.

4. That when fibrosis is found the men be given surface employment.

5. That the use of the dry drill be discontinued.

6. That all surfaces be kept constantly wet.

7. That provision be made for thorough ventilation.

Though the occurrence of miners' phthisis is proven, it does not appear to be a serious problem at present. This is doubtless due principally to the fact that few of the men remain strictly at the one type of work, but move about from mine to mine or frequently leave the mines for other occupations. As the mining industry develops to larger proportions in this camp this factor will lessen and the rate of incidence tend to increase. The incidence will be lessened by the discontinuance of the dry drill which has been effected by the Mines Inspection staff of the province. Further, through their efforts the large mines are now kept sufficiently wet and have provision for adequate ventilation.

Dr. Hall: The worst cases, however, are those that do not work underground. The men who carry the ore out on the car and dump it are those who get into more trouble than anybody else. As to tuberculosis, a great mnay of these men working underground cough more and more and run into tuberculosis. Their lungs are more susceptible to tuberculosis in later years.

Dr. Otis: For example, as Dr. Jarvis of Barre, Vermont, has shown, in a stone cutter who had followed this occupation for only a limited time, the x-ray plate showed a considerable amount of mottling or density while he was engaged in stone cutting, but if he abondoned this occupation for a while this mottling or density cleared up. Then again, as Dr. Jarvis has demonstrated, stone cutters may die of pure pneumoconiosis without the superimposition of tuberculosis.

Dr. Elliott: The diagnosis was made from physical examination, sputum examination and the history, but no diagnosis was made until the x-ray plates had been studied. The case of silicosis showed the characteristics, generally diffused with fibrosis. The mines are working down to 1200 ft. level. Five thousand feet is the limit.

A CASE OF HYDRO-PNEUMO-PERICARDIUM IN A TUBERCULOUS INDIVIDUAL DURING AN ATTACK OF TYPHOID FEVER.

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Hydro-pneumo-pericardium is, as all are aware, a rarity of medicine. So rare is this condition that it might almost be said to be a curiosity. Many engaged throughout long years in the active practice of medicine have not encountered a single instance. Medical literature contains accounts of approximately only forty-four cases. Many of these were summarized by Walter B. James[1] in 1904 in an article describing a case of his own and mentioning thirty-seven other reports. Since then Cowan, Harrington and Riddle[2] in 1913 reported five additional cases, and in 1918 Charles Stewart[3] reported another.

From the reports of James, Cowan, Harrington and Riddle, the following interesting facts are extracted: In thirty-four instances there was an opening from the pericardium to some other viscus. This opening was largely due to trauma. In nine the puncture came from without. Among the direct causes may be mentioned the swallowing of false teeth, "sword swallowing," bone swallowing, fracture of ribs or sternum, paracentesis of pleura, cancer of esophagus, softening of tuberculous focus (lymphnode), emphysema, pneumonia, gangrene, empyema, hepatic abscess, gastric ulcer, appendicitis, facial erysipelas with "sore throat," and pericarditis.

In only a few cases was an accompanying pericarditis absent. Infection with effusion, which later became perulant, occurred in the majority.

The fluoroscope or x-ray was used for diagnosis in only four of the cases. This was due mostly to the fact that most of the cases occurred before the discovery and use of the x-rays.

Physical signs led to a correct diagnosis during life in thirty-seven instances. Usually the apex impulse disappears, the area of cardiac dullness is replaced by tympany, the heart sounds become

distant and, when effusion develops, a "rhythmic, splashing, churning, friction" sound (James) "Le bruit de la roule hydraulique" (water wheel sound) is noticed. The tympanitic note is replaced by fluid dullness and vice versa upon change of position of the patient.

Symptomatically there is usually cyanosis, precordial oppression and syncope. In our case the heart sounds were audible to the patient and to anyone twelve inches from him, without the use of the stethoscope.

The condition is by no means always fatal as sixteen of forty-five cases (forty-four+ours) recovered (36%).

Our case occurred at the Trudeau Sanatorium during an epidemic of typhoid fever which has been reported elsewhere[1]. Unfortunately no x-ray picture was taken because it was thought that the patient was too ill to risk moving to the x-ray department in another building. No portable apparatus was then available. Following is the detailed history of the case:

Case No. 4669. Mr. C. W. H.

Admitted March 31, 1917, male, aged 26, single, bookkeeper. Family history negative for tuberculosis, cancer, heart and kidney lesions. No history of exposure to tuberculosis. In October, 1916, symptoms of pulmonary tuberculosis were noticed followed in December, 1916, by an hemoptysis of an ounce, which was later repeated and followed by streaked sputum for two weeks.

Examination at Trudeau revealed tuberculosis affecting the right lung in the upper two-thirds and the left lung to the clavicle. Tubercle bacilli were found in the sputum. The x-ray revealed no unusual enlargements at either hilus.

While undergoing treatment nothing unusual occurred. After four months the patient was discharged showing definite improvement in his condition. He was a quiescent case. He was then employed at the sanatorium as bookkeeper. He remained well for another month, when on August 24, 1917, he complained of headache and malaise. Temperature on this day normal. August 25th the temperature gradually rose, "biliousness," nausea, etc., occurred and as there were other cases of typhoid at the sanatorium at that time, a provisional diagnosis of typhoid was made, and later confirmed by finding the typhoid bacilli in the blood and feces. The

patient was never very toxic, the spleen was never enlarged. Fever subsided in twenty-two days and the patient went on to complete recovery.

On September 5th, 1917, the eighth day of the typhoid, the patient complained of pain in the left chest and shoulder and that he could hear his heart beat when he turned on his left side. Examination of the precordia showed very faint heart sounds in the recumbent position. The area of cardiac dullness was not increased. When the patient turned on his left side or took a semi-recumbent position, a distinct rough pericardial friction rub was heard. In two days cardiac dullness was replaced by resonance and a distinct pericardial splash was heard. The friction rub was still present. On the third day hyperesonance replaced dullness when the patient was recumbent. When lying on his right side dullness was present to the left sternal margin. Friction sound and gurgle were still present. On the fourth day the patient said he could feel the cardiac splash on changing his position. The heart sounds on this day in the third and fourth interspaces were heard chiefly during inspiration. During expiration they almost disappeared. Along the right border of the heart the sounds were loud to the nipple line. Dullness was present to the right of the sternum when the patient was on his right side. When he turned on his left side a splash was heard, followed later by a friction rub. The patient was still conscious of the friction rub.

On the sixth day there was no palpable or visible impulse over the precordium. The normal area of cardiac dullness was replaced by hyperesonance. With the patient on his back the heart sounds were indisinct. The first sound was heard best in the third interspace three cm. to the right of the sternum. The aortic second sound was heard fairly dsitinctly at the base. The second pulmonic sound could not be heard. With the patient on his left side a distinct to and fro friction sound was heard in the fifth interspace seven cm. from the mid-sternal line. This sound was followed by a splashing sound which was accentuated by shaking the patient and made audible to the ear at a distance of six to eight cm. from the chest. The area of cardiac dullness was not changed.

With the patient on the right side there was a definite impulse in the right third interspace. No shock or thrill was felt. The area

of cardiac dullness was not changed but dullness was intensified. Heart sounds at the base were more distinct. The first sound was audible in the fifth interspace seven cm. from the mid-sternal line.

On the tenth day the patient complained of a "heart knock sound" when lying on his back and while holding his breath at the end of expiration. Auscultation revealed a loud liquid knocking sound over the precordium, which disappeared when the patient resumed breathing. On the thirteenth day the "knock" and "splash" became less audible. On the fourteenth day relative cardiac dullness returned to normal and the heart sounds were distinctly heard. Breath sounds in the lower left axilla, which were somewhat distant from the outset, were now more distinctly heard. On the nineteenth day a palpable impulse was present in the left sixth interspace. Absolute cardiac dullness was restored. The heart sounds were normal.

At no time during this illness did the patient have cardiac or respiratory distress. Cyanosis was not present. For the typhoid the patient was given a modified Coleman high caloric diet with fluids sufficient to produce free elimination of urine. For the pericardial condition an ice bag was used over the heart from the first to fourteenth day. Recovery was uneventful, the pulmonary tuberculosis was not aggravated and the patient was discharged October 24th (two months after the onset of typhoid and forty days after onset of hydro-pneumo-pericardium). He has since remained well (1922).

REFERENCES.

1. Pneumopericardium. James, Walter B. Trans. Assn. Amer. Phys., 1904, XIX-351-363.
2. On Pneumopericardium. Cowan, John; Harrington, Arch. W.; Riddle, J. R. Quart. J. Med., 1913-14, VII-165-171.
3. Pneumopericardium Following Paracentisis. Report of a Case. Stewart, Chas. E. J. Amer. Med. Assn., 1918. I-LXX, 1222.
4. A Study of the Effects of Typhoid Fever and Antityphoid Immunization on Pulmonary Tuberculosis. History of a Typhoid Fever Epidemic at the Trudeau Sanatorium. Brown, Lawrason; Heise, F. H.; Petroff, S. A.; Wilson, Geo. E. Amer. Rev. Tubers., 1919, II, 12, 717-733.

THE PHYSICAL EXAMINATION OF GRADUATE STUDENTS.

By WILLIAM G. SCHAUFFLER, M.D.,
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Over sixty years ago there was introduced at Amherst College a regular system of physical examinations of all men entering college. Careful records were kept and comparisons made of each student's condition at the beginning of Freshman year with his condition during the succeeding years of college life. These examinations proved to be of such value that practically all first class colleges and universities have followed the example set by Amherst, and today no one questions the great importance of this means of keeping the student body fit physically, nor of the influence it exerts on the student's mentality.

During my service as sanitary inspector of a division in the war, I was struck with the fact that so many young clergymen of draft age were unable to pass the physical examinations for the army, and were obliged to seek service in the Y. M. C. A. or other welfare organizations. On my return from overseas I settled in Princeton, New Jersey, and in the fall of 1920 suggested to the faculty of the Princeton Theological Seminary, one of the oldest institutions of the kind in the country, the advisability of giving to all its students the opportunity of undergoing a thorough physical examination. The plan was accepted and I drew up an examination blank, a copy of which is herewith presented.

The examinations were optional and 117 men, or about two-thirds of the students, availed themselves of the opportunity of finding out just where they stood physically. It was a striking fact that, of the first fifty men examined, one passed a perfect examination, two were in good physical condition and forty-seven men were deficient in one or more particulars. The principal defects noted were faulty eyesight, bad tonsils, underweight, too high or too low blood pressure and varying degrees of anæmia as shown by low

hæmoglobin count. Very many had defective teeth. These conditions were particularly noticeable among the first year students, many of whom had seen service in the army. I made it a point to tell each man wherein he was deficient and advise him how to overcome the difficulty. For the most part the advice was well received, and many of the deficiencies were quickly remedied.

In the fall of 1921 I started again to examine the men, and 156 presented themselves. Those who had been examined during the previous year were particularly interested in comparing their new records with the old ones, and it was gratifying to see a marked improvement in many cases.

During the past school year I have examined 197 men, practically the entire student body, with the exception of about thirty post graduate and partial students, many of them men considerably older than the average.

During the three years I have examined forty-one men three times, ninety men twice and 183 men once.

Of the forty-one men who have been examined in three successive years there has been an increase in weight in twenty-six cases, improved blood pressure in sixteen cases, improved hæmoglobin in sixteen cases, and all but five of the men have shown generally improved conditions, in many cases due to removal of bad teeth and tonsils and regulation of diet and exercise.

I will not burden you with further statistics, some of which might be of interest, as my purpose is merely to bring to your attention what can be done for post graduate students in our seminaries and other graduate schools by paying more attention to their physical condition on entering and during their course of study. I have communicated with twenty-one theological seminaries, asking whether any physical examinations are made and whether there is any supervision over exercise, etc. Twelve answers have been receivd. In four institutions, apart from Princeton, examinations are made, in one a certificate of health is required for entrance, and in five there is opportunity given for exercise. It will thus be seen that in the great majority of graduate schools of this character the physical fitness of the students is not considered, nor are adequate means taken to preserve and improve their health during their course of

study. I have no doubt that the same holds true to an even greater degree in our medical and law schools, though I have no statistics to prove it.

In addition to making the physical examinations, I have given a series of lectures to the student body on Personal Hygiene, Public Hygiene, Social Hygiene and Mental Hygiene. These are subjects concerning which there is a lamentable ignorance, even among college men, and I have found that instruction along these lines is eagerly welcomed by men who are preparing to serve their fellow beings in the ministry.

My purpose in presenting to the Society this brief report is the hope that it may stimulate you to suggest and encourage similar work in post graduate institutions all over the country, and thus help to improve our great body of professional men.

PREVENTIVE MEDICINE IN INDUSTRY.

By CHARLES W. CRANKSHAW, M.D.,
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The practice of hygiene and sanitation in business has increased considerably during the past few years, and the solution of hygiene problems in the laboratory has contributed in no little way to the means now used for the protection of those employed in or about the shop, factory, or office of great or small industrial establishments.

The social service worker is helping to solve some of these problems, and greater service can be rendered if a visiting nurse is employed to visit employees at their homes or in the hospital when they are sick or injured. The social service department of the Massachusetts General Hospital serves as a striking example of what we mean by real social service work, and is rendering excellent service, counting mightily for efficiency. In this way, prevention, and not cure, plays the title rôle.

Preventive medicine is of great value commercially, and it is true conservation of time, money, and physical and mental energy. In industry all education must be turned to practical account. Many of the large and small industrial establishments look after the health and welfare of their employees. As to the hygiene provisions of a great insurance company like "The Prudential" where over five thousand employees in the home office are busily engaged in the conduct of the business, it is quite essential, in order to get the highest efficiency, that everything possible be done to safeguard the health and conserve the lives of those employed in or about the five large buildings, composing the group known as the Prudential home office.

The greatest attention, then, should be given to the care and welfare of the most wonderful mechanism ever created — the human body. Some of us try to exercise great care as to the hygiene of our bodies, while others, who think they know better, persist in doing things to the detriment of the mechanism, and it is little wonder that their physical and mental powers become impaired and inefficient

long before they should. Nature is patient and long-suffering, but she requires a final adjustment of her account. To begin with, all applicants for clerical positions with the Prudential are required to take mental tests and are also subjected to a medical examination. Applicants for positions in the printing department and bindery, commissary department, engine room, electricians, machinists, elevator men, porters, etc., are required to pass a medical examination before entering the service. In the commissary department, in addition to the examination on entering the service, an examination is made every six months of all those handling the food, dishes, or linen. This includes everybody, from the manager, assistant and chef to the dish-washers. We pay special attention to diseases of the lungs, heart, eye, ear, nose, throat, mouth, teeth, skin, etc. Swabs are taken of the nose and throat and a specimen of blood is also sent to the city laboratory, thus complying with the requirement of the city health department. The urinalysis are made under the supervision of the Director of the Prudential Laboratory.

We will refer to the problems of hygiene, safety, and welfare of the employees, worked out and put into operation by the Prudential in the Home Office, under the following headings:

Ventilation
Lighting
Heating
Drinking water
Dust removal
Flies, etc.
Rubbish disposal
Washing facilities and toilet
Arrangements for soap, towels, etc.
Lockers
Drying room for clothing
Commissary department including kitchen, refrigerators, butcher shop, dishwashing rooms, bread baking, ice cream and serving rooms, dining rooms, cafeterias and garbage disposal.
Safeguarding machinery
First aid hospital or infirmary work, graduate nurses for the infirmary and visiting service, cases referred to rest, and recreation rooms, cases for milk with ovaltine, or egg.
Service allowances, service retirement, insurance and sanatorium allowances.
Library
Office hours, vacation, and special days
Telephone exchange
Rest and recreation
Concerts, etc., also
Field and athletic games.

Ventilation.

Great attention is being given to the matter of ventilation, and in some parts of the building the air is washed and pumped into the rooms; others have a ventilator connected with each window that can be opened and shut at will, and these are connected with the radiator system, so that in winter the air can be warmed as it comes into the room. Electric fans are installed throughout the buildings, and these, in connection with ventilating apparatus installed, and the fact that the buildings are well supplied with windows, insure to the employees an abundant supply of fresh air. The air in each department is sure to be swept clean at intervals throughout the day as the managers of the different departments and divisions see to it that the windows and ventilating devices are properly adjusted, thus ensuring rooms that are well ventilated. There may be isolated parts of the buildings where it may be necessary to pump in air, and in one or two such places a ventilating system has been installed at some expense, but, when it comes down strictly to good ventilation, it is my opinion that an abundant supply from the outside, can be secured through the opened windows, as the old-fashioned idea of opening the windows, cannot be excelled by any system of washed air, afterwards pumped into certain rooms or departments, unless such rooms are some distance from outside walls. In the large vaults for filing papers, our machine shops and one or two parts of the building, where it is impossible to have a direct connection with the outside air, machines have been installed to pump in fresh air, thus insuring an abundant supply to those employed in such departments. As I said before, the problem of ventilation is continually being studied, and wherever the system can be improved, this is being done. From our present knowledge we know room temperature should be kept as near 68 as possible and the air kept in motion.

Lighting.

The buildings are all well equipped with large windows, and there are very few parts of the buildings, in which it is necessary for artificial light to be used, alone, throughout the day. Proper window shades are hung to each window, so that the light can be properly adjusted to the degree of sunlight outside. In case artificial light has

to be used, each desk is properly provided with an electric light, which is held up from an arm which can be swung to the right or left according to the position of the one sitting at the desk. This light can be lowered or raised, as the case may be, and the electric bulb is properly screened with a suitable green shade. In addition, the large blotters which are placed on each desk are of a green or blue color, and these serve to rest the eye to a great extent. The indirect lighting system, where necessary, is being gradually installed throughout the buildings, and special attention is given to the lighting of the filing department, vaults, etc.

Heating.

The buildings throughout are heated with steam, and some of the rooms have thermostats or automatic adjusters to regulate the temperature.

Drinking Water.

One of the most important things for the employer to consider is that of the drinking water supply, and, as we found from the reports received from the city laboratory that it was not safe to use the water from our driven wells for drinking purposes, this supply has not been used except for mechanical and toilet purposes. We use the water which comes from the city mains. This is tested frequently by the city bacteriologist, and as we are constantly in touch with the board of health chemist or city bacteriologist, we are assured that the water used for drinking is safe. In fact, the bacteria content of our drinking water is lower than that of the supply direct from the city mains, for the reason that we have a filtering plant, and the water passes through the filters and then through a cooling process, before it enters the drinking fountains, which are installed throughout the buildings. A recent laboratory report showed twenty bacteria per cubic centimeter before filtering and five bacteria per cubic centimeter after passing through our filters. Some of the samples after filtering have been sterile. We have never had colon bacilli in any of the samples. The filters are used and cleaned alternately. High-pressure steam is used for cleansing when necessary. Each employee has his own drinking glass, and consequently the chances of infection with any contagious disease, by means of the

secretions of the mouth or throat of others are very small. Water is so necessary to the various activities of the organs of the body, aiding secretions and excretions, food absorption and carrying away waste products, regulating body temperature, etc., that, to encourage the drinking of sufficient water to help these various functions of the body, it is quite necessary that we know the quality of the water supply, and can assure the employee that it may be used as often as is necessary for drinking purposes, with no fear of infection. It is a comfort to know that, as far as bacteria content or colon bacilli are concerned, the water is perfectly safe. In this way, one can readily see that fatigue poisons are diminished, and we attempt to prevent diseases that may be caused by impure water, or the use of the common drinking cup. It is easy to understand that efficiency is increased where wholesome drinking water is supplied, and without much study an economist can see the financial value of this side of the question.

Removal of Dust.

In regard to the removal of dust, absolutely no cleaning is done during office hours, and, as a vacuum service is installed throughout the buildings, the dust from the floors and rugs which is taken up in this manner is all centralized in one container in the basement of the building. A movable cleaner of this sort is now used for books, paper-files, etc. The rubber coverings which are used in many of the corridors and aisleways for deadening the sound, thus preventing unnecessary noise, by those having to walk in, through, or to a department, have a sweeping compound distributed over their surface before they are swept. This is used on parts of the floor also. The use of the common duster is discouraged as far as possible, and it is used only where it is impossible to use other means of cleaning which would be practicable. When such dusting of necessity has to be done, the windows are thrown wide open and the electric fans started; no persons but the cleaners are about, and their exposure is slight, as the dust is quickly carried out of the windows.

Flies and Insects.

As to flies and insects of any sort, we are especially interested in keeping these out of the infirmary and the commissary department,

and by eternal vigilance they are kept down in all parts of the buildings by keeping out rubbish and dirt. In the Infirmary the screens are kept on the windows throughout the year, and in the Commissary Department from April to December.

Rubbish Disposal.

Surgical dressings, wooden tongue depressors which have been used, and other material of like nature from the Infirmary are all subjected to incineration. The basements of the buildings are kept entirely free from all rubbish, which is carted away each day, and the walls are thoroughly whitewashed or painted throughout the corridors and rooms in the basement of the building. Most of the basement corridors are painted and the floors are concrete. Notices are posted forbidding spitting and smoking.

Lavatories.

As to washing facilities and toilet arrangements, all the washrooms and toilets have marble floors and part of the side walls are marble. These are subjected to a thorough cleansing every day, and at least once a week to a thorough scrubbing with an antiseptic solution containing soap. As we do not advise the use of the water in the toilet rooms for drinking purposes, because this water comes from our driven wells, proper notices to this effect are posted, and also notices with reference to the continual flushing of the toilets. Disinfectant is used systematically in each toilet basin throughout the buildings. Soap is provided for use in each toilet room. In some rooms liquid soap is used from a container. The roller towel has been replaced with individual towels of linen or paper, according to the needs. Several years ago the company supplied an individual drinking glass to each employee, and as employees enter the service they are given a similar glass. Such provisions lessens the chances of skin infection or infection by the secretions of the mouth, throat, or nose. A matron is constantly going about looking after sanitary matters in the women's toilets. These have been supplied with machines for sanitary needs. Practically all of the toilet rooms are provided with an electric fan.

Lockers.

Each employee is supplied with a suitable locker, which will hold the necessary articles of overclothing, and they contain the proper arrangements for umbrellas, overshoes, etc. The lockers that have been installed during the past five years are metal.

Drying Room for Clothing.

A drying room for clothes has been installed, so that on very inclement days the clerks need not run any risk of catching cold on account of getting wet. Rooms for rest are at their disposal and to the females, skirts, slippers and one-piece aprons are supplied, and in a short time their clothes are returned to them perfectly dry. If any condition tending to impairment of health is noted as the result of exposure in coming to the office on such days, the clerk or employee is sent home at once. Rubber boots, coats, and hats are supplied to a few men who are required to be on duty, exposed to the weather, on very stormy days, such as street officers and porters.

Telephone Exchange.

The young ladies who do this work have, in addition to their noon lunch hour, an additional twenty minutes' rest period both in the forenoon and in the afternoon. They have a well appointed rest room adjoining the telephone exchange and their surroundings and comfort are being constantly looked after, as their work is of an exacting nature and requires the eye and ear to be constantly on the alert.

Commissary Department.

In the Commissary Department, which includes the kitchen, butcher shop, large regfrigerators, bread baking, ice cream making, serving rooms, also three large cafeterias and three smaller serving rooms and dishwashing rooms, great care has been taken to install the latest improved apparatus for washing the dishes and kitchen utensils. The floors and walls are kept scrupulously clean. All dusting necessary is done with moist cloths, and the table linen receives the utmost care and attention. The refrigerators are cleaned

several times daily, and kept scrupulously clean at all times. The windows of the Commissary Department, cafeterias, dining rooms, etc., are all screened. Two large cafeterias on the tenth and eleventh floors of the North building, provide a hot lunch at noon consisting of soup, meat, vegetables, milk, tea, coffee, dessert, etc. for 3,100 girls and women; a large cafeteria on the tenth floor of the Main building provides the same for 1,500 boys and men. The three smaller dining rooms take care of the remainder of the 5,000 employees, including the senior and junior officers, managers and assistants. The garbage from the kitchen, cafeterias, dining rooms, etc., is disposed of promptly every day. It is not subjected to incineration, but is carted away daily, the cans cleaned as soon as they are emptied and afterward subjected to a high pressure of steam.

Milk and Food Supply.

The milk and food supplied to the employees are matters which receive great attention. The milk is received from two dairies which have reliable reputations for having the latest devices for cleaning and pasteurizing. This milk is examined bacteriologically at intervals in one of the largest scientific laboratories in New York City, as well as local laboratories, and we endeavor to make frequent inspections of this milk, which is classed as grade A pasteurized. We are assured that all of the cows are tuberculin-tested, and we feel perfectly safe that no disease can result from this milk. The meats, poultry, and all food supplies to be served in the cafeterias and dining rooms are selected with great care and put into proper refrigerators as soon as received. This is quite necessary when you think of the number of people served by the Commissary Department daily, and if you ever have the opportunity to visit the Prudential Commissary Department, you will note that the latest and up-to-date practical devices, utensils, etc., have been installed.

Safeguards, Machinery, Etc.

In the engine room, printing and bindery departments, and every place where machinery, electrical devices, etc., of any sort are used, they are all properly safeguarded. Either the protectors have been

on the devices when purchased or attached to certain parts of the machinery after they have been installed. In this way accidents have been reduced to a minimum. The gates of all the elevators have proper safeguards and devices for the levers which open and close the doors. For absolute safety, no elevator man is allowed to leave his car during the time he is on duty, except by proper arrangement with the starter. The door of the car cannot be opened until the car is brought to a full stop, and the car cannot be started until the door is closed, as the elevators have electric appliances which regulate the movements of the car. The window washers are all supplied with safety belts, and under no consideration are they allowed to wash or clean windows, etc., without using these appliances. The fire captain maintains fire drills, and the necessary appliances in case of fire are placed throughout the buildings. Through the real estate department, efficiency committee, office supervisor's, etc., problems along these lines which come up from time to time are given thought, study, and consideration, and our records show that the number of possible accidents have thereby been reduced.

Infirmary Work.

All cases of sickness or accident are sent to the Infirmary immediately. In order to facilitate this work, the company about three years ago set aside more room to be commensurate with the growing importance of the department. At present we have nine large rooms, comprising about two-thirds of the floor space of the front part of the North building, on the ninth floor, where we have excellent light, plenty of fresh air, and the rest rooms being on the court side, are quiet and restful. These rooms comprise a waiting room which can be easily approached from both ends of the building through the corridor which is well lighted and delightfully arranged. A young woman with the proper attainments, social, etc., has been selected as special clerk and she has a desk near the double door of the waiting room and can receive the patients easily and direct them to the proper room where the nurse on duty in such room can minister to them as far as she is able to do. This clerk looks after the infirmary records also. On one side of the corridor is the treatment room for first-aid, etc.; a very large rest room for the female employees, and

adjoining this, one for the male. Everything for their comfort has been installed. Adjoining the waiting room on the opposite side of the corridor is the nurses' room where all patients needing examination are first interviewed by the nurse. Sometimes two nurses are on duty in this room. They can make preliminary eye tests, record the pulse, blood pressure, make arrangements for urinalysis of the females, take height, weight, etc. The room is large enough so that it can be used privately for the nurses when necessary. Next to this is a well appointed room used by the associate physician for consultation, periodic examination of employees, disability cases, etc. The physician in charge has a well equipped office immediately adjoining the examination room, and next to his office is the stenographer's office where all of the disability, service, retirement, sanatorium records, etc., are kept. Adjoining this is the Home Office disability section where several clerks are required to tabulate the cases and keep the proper records for the Service Allowances Committee. The Infirmary staff at present consists of a physician in charge, associate physician, five graduate registered nurses, stenographer having secretarial duties and a special clerk. Two of the nurses are on duty a greater part of the time as visiting nurses. In a case of very severe injury we arrange to have such a case taken immediately to the hospital to which the injured may desire to go. In cases of illness, if it is plainly seen that the patient is not going to get better speedily, arrangements are made to take such cases home or to a hospital, as they so desire. Any case which has not recovered sufficiently to go home unaided is accompanied by a nurse or some one from the Home Office, and, if necessary, a private auto conveyance is arranged for, and in extreme necessity we have arranged for a private auto ambulance, so that the case can be dispatched at short notice. Every attention, as far as possible, is given to the slightest detail for the comfort of those who are sick or sustain any injury, and the fact that their welfare is looked after so carefully along this line is greatly appreciated.

Occasionally cases come before us which for some reason are not able to easily assimilate their food, and other cases, which have completely convalesced after surgical operation or serious illness in their homes or at the hospital, are strong enough and desirous of resuming

duty, but need additional nourishment. If they so desire, it is arranged that, on the order of one of the physicians or nurses, they may go to the rest room at 10.30 in the forenoon and 2.30 in the afternoon for milk and ovaltine, or milk alone, as they desire. One of the nurses is detailed to give personal attention to these cases daily, and by frequent observations she can note the weight, general appearance, etc. The results of these cases are extremly gratifying. Some have gained as much as ten or twelve pounds in weight, and the quality of their blood is better. There is the joy of feeling well, not being fatigued nearly so easily, and their efficiency has been greatly increased thereby. In addition to the extra nourishment afforded, there is, of course, the additional rest, and the fatigue toxins do not get a chance to diminish the vitality of such cases. We can thus see that the Infirmary is a clearing-house for practically all cases of illness or injury, and acts in the same capacity in reference to quarantinable diseases. Since the disability plan has been in operation, such cases naturally are taken care of by this department. All employees in the office who have any infectious or contagious diseases at home are to report same to the heads of their department immediately, and they are in turn referred to the Infirmary, so that we can determine whether they should absent themselves from the office. In this way we are doing much to conserve the health and lives of those who are conducting the business at the Home Office. The visiting nurse carries good cheer and a kind word to those who are absent on account of disability. She is able to advise in many cases what is the best course of procedure, so that they will soon be restored to health, and in addition she is encouraged by the physician in charge to keep him in touch with any complications or data which will aid him in gving proper consideration to any given case. In our infirmary and disability work we have the most cordial relations with the hospitals and sanatoriums, and also with the attending physicians, specialists, and dentists. In many cases the family physician asks us to coöperate with him, in order to effect a cure. When we find cases of incipient tuberculosis of the lungs, they are referred immediately to their family physician, and in turn we are asked to coöperate in any sanatorium or other treatment. Our experience in handling this class of cases has been very satisfactory, and today a number of cases

which at one time were those of open or active tuberculosis have had the disease arrested and been restored to health, so that they could safely return to duty. At present we send our women and girls of the Home Office who need sanatoria treatment, to Stony Wold Sanatorium, Lake Kushaqua, Adirondacks, N. Y. Those of the men or boys who should go to a sanatorium are referred to Loomis or any suitable sanatorium to which they desire to go. The field men are placed in the nearest suitable sanatorium, according to whether they live in the United States or Canada. The following figures for the year 1922 will give some idea of the work done in the Infirmary.

	1922
Sickness cases	6,960
Accidents on duty (many of these minor cases)	572
Accidents off duty (many of these minor cases)	670
Total cases treated	8,202
Cases referred to visiting nurse	1,564
Visits made by visiting nurse	1,886
Grand total of visits, examinations, disability cases, treatments, etc.	14,881

Percentage returned to duty after receiving treatment at the Infirmary since 1913 (these years include the flu epidemic and World War):

1913	85.5	1918	82.7
1914	83.3	1919	82.3
1915	86.3	1920	82.2
1916	86.0	1921	85.3
1917	87.1	1922	85.5

Our field representatives in the United States and Canada number over 15,000. We have excellent coöperation with them as well as the Home Office force. In our tubercular cases where sanatorium treatment is necessary, in addition to their being paid a service allowance they are given a sanatorium allowance for a period of twenty-six weeks. The majority of our cases do very well in that time. All cases, Field and Home Office, have their expenses paid from the time they leave home for the sanatorium till their return. They should do well if we can arrange to get them to the sanatorium at an early

date as they are relieved from mental anxiety and worry knowing that their expenses at the sanatorium are taken care of by the company each month and their check for service allowances is sent to their home to take care of the expense at that end, if they so request. This of course would apply more forcibly to a man with a family or one having financial responsibilities at home. Needless to say, this arrangement is greatly appreciated by those needing sanatorium treatment.

Disability, Service Allowances, Service Retirement, Insurance and Sanatorium Allowances.

In case employees are disabled, they are paid according to their length of service. No contribution is required from them, but their regular compensation is continued as usual on account of disability, according to the plan which was announced by the president of the company in December, 1913. As to service retirement, this is also arranged for according to age and length of service. Pamphlets have been printed giving the entire plan of the disability, service allowances, service retirement, insurance and sanatorium allowances in detail.

The Prudential Old Guard.

This is an organization within the Prudential membership, in which, in the words of its founder is "based upon a long and continuous and honorable connection with the company." It was formed in 1868. A Prudential employee who has served five years enters into Class A, receiving as a mark of membership a certificate and bronze emblem. Ten years' service (Class B) a silver emblem, fifteen years' service (Class C) a gold emblem, and each subsequent class is fittingly rcognized. The classes now include H, indicative of forty years' service.

Insurance on Lives of Field and Home Office Employees.

On July 17, 1916, the president of the company announced to the industrial field staff and the Home Office force that the company would insure the lives of its employees without any cost to them, whether on the active or on the retired list, for the amounts indicated

in the following schedule, and that on the death of any such employee while in the service of the company there will be paid, in one or more installments, to the person or persons appearing to the company to be equitably entitled thereto from $500 to $2,000, according to the length of service.

Except in the cases of employees on the retired list, the foregoing applies only to persons whose whole time is given to the service of the company and does not apply to the officers or directors.

Library.

During the past year the Dryden Memorial Library enjoyed its most successful year. It shares the interest of the employees of the home office with the two recreation rooms, and on rainy days there is scarcely a vacant chair during the noon hour. About two thousand employees are now enrolled as borrowers of books and magazines, and there were thirty-two thousand books in circulation during the past year. The care of the vacation bureau is at present a part of the library work. This bureau has been of service in many ways to a part of the home office force.

Office Hours and Vacations.

The office hours during each working day are from 8.30 to 12 and 1 to 4. Saturday is a half-holiday during the entire year. Vacations are arranged for according to length of service, and only in a very few instances — those of beginners — are vacations less than two weeks. Some of the older employees enjoy as much as four weeks. In addition, a special day is granted every thirteen weeks for promptness, punctuality, special care, and attention to duty.

Rest and Recreation.

As to rest and recreation, the general welfare of the employees is being considered, and, in addition to those who have the privilege of going to the rest or recreation rooms, it is desired as far as possible that all employees should absent themselves from their desks or departments during the entire lunch hour, as they have one-half hour to eat and a one-half hour for recreation. This gives them

sufficient time for luncheon and a short walk in the fresh air and sunshine, or for any other diversion which may be of benefit to them. Each working day the clerks enjoy a recess at 10.30 A.M. and 2.30 P.M., so that no period during the day is longer than one and one-half hours, excepting the first period, from 8.30 to 10.30 A.M. A large recreation and game room has been fitted up for and is thoroughly enjoyed by the men. The women's recreation room is well equipped, and the many delightful appointments make the room very popular. Part of this room, which is 144 feet long, is reserved for dancing, and there is excellent supervision by the House Mother or Welfare Director. In the remainder of the space calisthenics, games, etc., may be indulged in. Rest and recreation are very important, as they tend greatly to reduce fatigue toxins, and efficiency is thereby increased.

Concerts and Entertainments.

Good use is made of the assembly hall, recreation room, etc., during the luncheon hour. There is a diversity of talent among the five thousand employees, and the concerts, movies and other entertainments are well attended. The musical director has organized a good band and orchestra; also an excellent chorus. The arts and crafts exhibition brings out an exceedingly creditable display of the handiwork of the employees, and their products of yard and garden are shown to advantage in the popular flower show.

Field and Athletic Games.

The Prudential athletic association has charge of all the athletic and recreative sports, and, in addition to tournaments, cross-country runs, athletic meets, and games which are held at different times during the year, has at least once a year a field day; and the entire force is given the privilege of enjoying this outing.

An additional day is also given in August for an excursion to the seashore. This association has as active members, about 90% of the home office force, and they are doing a great deal to help themselves along the lines of hygiene and health, and are receiving the active coöperation of the president and other officers of the company.

I have attempted in a very hurried manner to give you an outline of some of the things which the executives and officers of the company have worked out for the general well-being and physical welfare of the employees. In view of the fact that all applicants for positions are subjected to a medical examination, and later to a periodic examination, eye examination and Wassermann test if necessary, we are able to safeguard the health of the employees to begin with, and as they are taught from time to time, by personal contact in the infirmary, by health articles which are printed in pamphlet form, and by other matters of hygienic interest which appear in the Home Office News, the young men and women are beginning to learn more about the laws to be observed as to the care of their bodies and what it means to have a sound mind in a sound body. It has been said that "life is not only to live, but to be well." How true this is and what joy there is in work, mental or physical, when we feel strong and well! Preventive medicine is, therefore, true conservation, and adds to the efficiency of those who are actively engaged in any kind of industry or line of business.

THE USE OF COLEY'S MIXED TOXINS IN THE TREATMENT OF CHRONIC ARTHRITIS.

By THOMAS KLEIN, M.D.,
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One need not look very far into the endless literature to realize fully the perfectly hopeless task of finding a suitable classification of the various forms of chronic arthritis. It is also not within the bounds of this short paper to enter into any lengthy discussion. Suffice it to say that the term chronic arthritis as here employed embodies those types which are variously classified as: Chronic non-suppurative arthritis; Rheumatoid arthritis; Chronic infectious arthritis; Chronic osteo arthritis or arthritis deformans. Gout or gouty arthropathies being a purely metabolic disease is not here considered. The same holds true to the arthropathies of nervous origin or those occurring in tabes dorsalis (Charcot joints) and in syringomyelia. The chronic hypertrophic osteo arthropathies occurring in the course of pulmonary tuberculosis, bronchiectasis, chronic bronchitis, malignant tumors of the lung, and various chronic cardiac conditions are again distinct and are not included in the above term chronic arthritis as here used. Syphilitic arthritis is again a more or less distant entity and is not here considered. Villous arthritis is probably a stage of chronic osteo arthritis and will be considered as such. Fibrositis and panniculitis will be described as a part of the chronic infectious process so frequently occurring in this type of case.

The vast majority of the remaining cases will fall into that group in which we are primarily interested. This group is characterized at the beginning by swelling of the smaller joints, both meta-carpo-phalangel and metatarso-phalangel. The process may be unilateral but is more often bilateral. In the early stages the joints gradually swell with little or no pain. This gives them the characteristic fusiform appearance. Upon examination they have a distinct doughy feeling. At times there is a questionable effusion into the joint. Pathologically this enlargement is due to swelling and hypertrophy.

of the synovial membrane and capsular ligament. Pain is only present upon pressure and active or passive motion. The disease progresses with definite periods of acute exacerbations, characterized by fever and its accompanying symptoms; increased pain and stiffness in joints and surrounding structures — frequently by focal manifestations as increased swelling and redness in the joint structures themselves. These periods are also associated with a slight increase in leucocyte count as compared during the stage of quiescence. In all respects it follows clinically the course of an infection. During this time the disease progresses upward involving the larger joints, namely the wrist, elbow, shoulders, ankle, knee, hip and the joints of the spine.

Soon after the beginning of the process which in itself is essentially an hypertrophy of the periarticular structures, we have an atrophic process set up:—The atrophy involves the muscles, subcutaneous fat and skin. These atrophic changes are undoubtedly due in many instances to a reflex atrophy. Vulpains idea, namely that impulses carried from irritated articular nerves alter the trophic activity of the cells in the anterior horns without causing a lesion, but sufficient to cause atrophy and weakness, is an extremely good one and is the best explanation offered. It is the glossy atrophic condition of the skin, atrophy of the subcutaneous fat and muscles plus their subsequent contractures which gives the characteristic deformity. The extensor groups of muscles always suffer greater damage than the flexors. Hence flexor contractures always predominate. This is explained by the fact that the nerves which supply the extensors also supply the joints themselves, consequently they bear the blunt of the irritation and the greatest atrophy.

Associated with this degenerative process is a fibrositis involving both the aponeurosis and the muscles themselves. In fleshy individuals the subcutaneous fat is also caught in the process in the form of a panniculitis. These two conditions account for a good deal of the patient's suffering and as we will see later form a very important part of the treatment.

The feeling that this type of case is always due to a chronic infection is fast gaining ground. Even though the causative organism is not found, the clinical course with its frequent, regular, acute

exacerbation associated with fever, increased swelling, pain and redness of the joints, increased leucocytes as compared with the quiescent period, is sufficient to establish this fact. Consequently, when such a case presents itself for treatment we immediately look for a focal infection. Teeth and tonsils correctly occupy the foreground as the most frequent sites of focal infection. Following these are the sinuses, gall-bladder, prostrate, infected ingrown toe-nails, ulceration along the intestinal tract, mediastinal lymph nodes, etc. The femal pelvis has not been found an important factor in our groups of cases. In one case now under treatment the mucous membrane of the mouth afforded repeated rich groups of a hemolytic streptococcus; otherwise no source of infection could be found. The prostate and seminal vesicles are frequently overlooked and when found as a source are too often looked upon as gonorrheal. The streptococcus may long persist without any gonococci being found. Non-surgical biliary drainage, done in an aseptic way offers many opportunities for the study of the gall tract for infection. Too often we overlook the bacteriology of the feces, as here is not an infrequent source of streptococcus infection.

Again we must remember that in the vast majority of cases, we are dealing with not one focus of infection but many. The eradication of a dental abscess or a pair of tonsils does not mean that we have removed all the source of infection. When one considers a mediastinal lymph node or an intestinal ulceration as a focus he will readily realize the difficulties in their removal. It is because of this fact and the repeated failure in not getting good results, that lead us to the utilization of a streptococcus vaccine. The vaccine also has further assets. It probably stimulates antibody formation to overcome any focus not eradicated and at the same time seems to aid in a causation of a retrograde process in the periarticular structures themselves.

During the past seven years Dr. Robert G. Torrey and myself have been using Coley's Mixed Toxins (a mixture of the streptococcus of erysipelitus and bacillus prodigiosus) in the treatment of this type of case with surprisingly good results. This vaccine was selected solely because of the potency or virulency of the streptococcus used (.01 cc. of the cultures being sufficient to kill a white mouse in

twenty-four hours). It is again easily obtainable in the market which is a distinct advantage to many men. The action of this vaccine is undoubtedly that of a high powered foreign protein but on the contrary after observing quite a series of cases one cannot help but wonder if it is not in some way a specific. It suggests the thought that the strain of streptococcus causing this disease is in some way related to the streptococcus of erysipelas. Thus far I have treated a series of twenty-one cases. Of these twenty-one cases, twelve have been cured, seven are still under treatment with marked improvement (diminution of pain, swelling of joints, decrease of ankylosis, acute exacerbation being lessened in frequency and severity, and finally abolished, with general improvement in health and gain in weight). Two cases stopped treatment because of severe focal and local reactions. The type of cases treated have been in the vast majority of cases far advanced. The earliest case treated was that of three months duration. The oldest case of fifteen years standing. The average length of duration previous to treatment was $3\frac{1}{4}$ years. The length of treatment varied equally as well. The earlier the case was started, the shorter was the duration of treatment. In the earliest case the vaccine was given over a period of three months. In the longest case the vaccine was carried over a period of fifteen months.

Mode of Treatment.

When the case presents itself the patient is studied thoroughly from all standpoints, especial attention being paid to focal infection, teeth, tonsils, sinuses, gall-bladder, stools, etc. If the patient presents gastro-intestinal symptoms fractional gastric analysis, feces examination and x-ray studies are made. The colonic stasis which is rather frequent in this type of case is corrected by diet and frequent colonic irrigation, using large quantities of water. The patient is usually undernourished, especially so in young people and I always encourage forced feeding; diets of special food have not proven satisfactory in any other way. For the past few years I have been making complete blood studies including complement fixation tests and the examination of urea, nitrogen, blood sugar, uric acid, creatinin and the chlorides. It has proven very discouraging with the exception

of an occasional high uric acid content. This I believe is of extreme importance in helping to differentiate some of these cases. One of my cases has a combination of a rheumatoid and gouty arthritis. She runs a rather persistent high blood uric acid. When the gall-bladder is under question the bacteriology of the bile is studied after asceptic transduodenal lavage. Surgery is only employed after a proved focus has been found. Needless to say that other various x-ray studies are made as indicated, including the joints themsleves. These patients are usually placed in hospitals for a short time during the starting of the vaccine. Rest is a very essential element in these cases and a few weeks in bed usually affords benefit. At the same time it accustoms the patient to the reaction of the vaccine.

In starting the vaccine it is very essential to start with a small dose, usually $\frac{1}{4}$ minim in 1 cc. salt solution or sterile water. This is given subcutaneously. Three types of reaction are experienced, focal, local and general. It has been my practice to tell these people that after the vaccine injection they will have pain in joints heretofore unknown to be involved. The pain in the joints is invariably increased after the first four or five injections. This thereafter will gradually be diminished and the period of freedom from pain will gradually be increased. At the site of injection a marked erythematous area develops. The fluid is quite irritating and at times you will think of abscess formation. This I have not experienced but for a week or more the hard indurated nodules persist. These patients are very hypersensitive to the vaccine and the dose must be increased very slowly. This hypersensitivity persists surprisingly throughout the course of treatment and varies with each individual. The largest dose of vaccine given was 10 minims and that after eight months treatment. In the case of fifteen years duration in which we obtained a beautiful result, I could never give over four minims without causing a general reaction. It is to be remembered that in these cases of long standing the patient is usually anemic and markedly undernourished. The pain has been severe and their nerve has been broken. Consequently it is important to try and avoid a general reaction. While this does not do the patient any harm, it is bad from a psychological standpoint. In addition they have been through so many hands and tried so many forms of treatment that

they are always dubious of any new form of treatment and especially so if they are to have more pain in the beginning.

The time interval of the injections varies from three to four days at the beginning, gradually being lengthened to five or six days, as the symptoms improve. It has been my rule to gage the time of injection and the size of the dose entirely upon the local reaction. If it be severe the same dose is repeated. At times it is necessary to repeat the same dose as many as four or five times before increasing the vaccine. Especially is this true as the larger doses are used. The vaccine is continued until the patient is clinically well. Unfortunately the time of stopping the vaccine is entirely emperical as I know of no way of telling when the infection has been entirely killed out.

The benefits derived are a diminution of pain plus a loosening up of the joints. The acute exacerbations which are so characteristic of the disease gradually diminish and are finally obliterated. It is remarkable to see the rapidity of diminution in the size of the joint. The patient through his own efforts will begin to move the joint and loosen it up as soon as the pain subsides. In the febrile cases the temperature gradually returns to the normal course. The patient's general health improves and he soon loses his toxic appearance. The gain in weight is quite remarkable. One patient who has been under treatment for the past seven months has gained forty-two pounds.

At the beginning of the treatment in addition to clearing up foci of infection, rest and vaccine therapy, the patient is given daily electric bakes to the joints involved; this quite frequently being a general body bake. Care is taken not to massage the joints or the atrophic surrounding structures until after the acute tenderness and swelling have subsided to a great extent. When the massage is started it is of the most gentle character, care being exerted not to traumatize an already diseased tissue. Manipulation, extension, deep massage and other mechanical devices are only resorted to when the patient is well toward recovery. The fibrositis in some cases persists and occasionally it is necessary to stretch the muscles under an anesthetic. Casts are applied but are removed each day for the bake, massage and manipulation.

In conclusion it must be said that the treatment of these cases

is as complex as their source of infection. The more thoroughly they are studied the more frequently I believe we will find definite evidence of streptococcus infection. Unfortunately the streptococcus does not lend itself to the formation of agglutinins or precipitins; consequently serological tests, so far have failed to give us any definite information. Coley's mixed toxins when used cautiously and over a prolonged period of time have in our hands given us a very satisfactory and pleasing result.

THERAPEUTIC VALUE OF COUNTER IRRITATION.

By HERBERT F. WILLIAMS, M.D.,

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It is interesting to trace the therapeutic measures of the ages. Recently we read of the discovery of the mummy of an Egyptian princess with "marks across the stomach" as if made by the moxa, probably essentially the same as is used upon occasion at the present time.

Whatever of value is gained by such method or by those more modern and perhaps less harsh, is explained on anatomical and especiallly vasor-motor provision. It must be a lack of knowledge of such an arrangement that not only keeps men from availing themselves of such assistance as proper counter-irritation affords, or perhaps permits them to give a quasi consent to the sometime inefficient and misdirected means in domestic use. John Hilton, F. R. S., in a series of lectures in the Royal College of Surgeons in 1861-62 said: "In order to bring in a comprehensive and definite form before you the fact which is so important on anatomical, physiological and pathological grounds, I will state is thus: The same trunks of nerves whose branches supply the groups of muscles moving a joint, furnish also a distribution of nerves to the skin over the insertion of the same muscles, and what more especially merits attention. The interior of the joints receives their nerves from the same sources."

This provision does not apply exclusively to joint articulation. The same arrangement is found in the cavities of the thorax and viscera and the skeletal structure. The late Professor W. H. Thompson in giving a definition of colds embodies the same idea. He says, "A cold is a circulatory and nutritious disturbance of an internal viscus or tissue, produced in the first instance by a reflex irritation from a cutaneous area with which the afflicted internal part is in nervous association." Such explanations from two eminent men were given years before the knowledge of the microbic assompaniment in inflammatory processes.

Professor Thomson foreshadowed such a discovery in the late sixties and early seventies in his lectures in the University of the City of New York. How nicely do these ideas fit and thus amplify our means of producing modification of acute or chronic processes, promote relief and hasten cure. We do not know all that may yet be known in the order and importance of the several alignments of disease, and from its varying manifestations it is difficult and often discouraging to determine what are the best means to combat it. As General Sherman said, "The logistics of war." Surely the attacking microbic force must find a vulnerable point of attack in order to make an effective assault. A defective defense in the subject of attack may be of recent or of long standing, preceding the hostile invasion. An acute bronchitis may seize a man in robust health, and immediately the conflict begins. His very strength may become a defective defense in an overabundant bronchial secretion which may become inspissated, most likely in early morning when sleep has quelled an expulsive cough. Pneumococci or their congeners are lurking in the arrested secretion yet innocent of the subjacent congestion due to the aerial stasis. The time is now urgent to thwart the microbes and their toxins in their proliferation. This more or less consistent infarct should be removed at once, and one of the most potent means is well directed counter-irritation. Personal experience is sometimes most enlightening, and I venture to give one relating to myself as more conclusive than any theory. Years ago, I was seized with a brisk bronchitis for a day or two; perhaps on the third morning I became conscious of a secretion which I could not dislodge. This was attended by an uneasiness, almost pain, about the congested area. In such a state I started on my professional rounds and my first patient was a smart, garrulous Irish woman in a similar plight. After some witty parleying on her part as I told her of my proposed method of relief, I commenced operations with a convenient book, and proceeded to counter-irritate and vibrate her lung with vigorous slaps. The intervals between, perhaps the increasing vigor by which the treatment was administered, were enlivened by a flow of rich Irish jocular invectives which were attended in a few seconds by an expulsive cough which loosened and dislodged the obstruction, then came a thankful "Be gorra you're a wizard." I

then told her of my similar predicament when in most appropriate Irish she urged the opportunity to reciprocate in the treatment. However, this was secured in an equally though more grotesque way. It was a sloppy day in March, some boys on an embankment were amusing themselves by "pegging" snowballs at pedestrians on the sidewalk. One directed at me struck a pool in front and bespoiled a new spring overcoat. This was an added jolt to my already perturbed state of feeling, and what perhaps might have been resented with a more dignified protest was exchanged for a choleric intent at chastisement. Accordingly I charged the miscreants, one of whom, more daring than his fellows, stood his ground and gave me a final shot just as I was within reach of him, then quickly jumped down the rear embankment the precipitancy of which I was unaware. My momentum was so great that I followed head first and landed on my side over the congested area of the lung, when quickly followed the loosened brother infarct of my Irish patient and her equally signal relief.

Here surely were two instances denied threatening microbic proliferation. We all know how frequently pneumonia develops in such a manner. Patients in similar conditions should be impressed with the necessity of making forcible expiratory effort, and instructed to open the glottis to force the expiratory muscles to make the act more effectual. Usually we get an admixture of a closed glottis cough and an open one; as I have said, this effort should be made in the morning. Yet at any time, as long as the symptoms persist and before febrile action begins, they should seek some one to vibrate the area in any available way.

So explicit have I been in giving such instructions that I find myself quoted as the author of the aphorism—"Cough saves more lives than it destroys."

While a member of a special class in physical diagnosis, of our lamented Alfred L. Loomis, he spoke to us of his personal experience with a bronchial infarct which as he said finally "drove him to the Adirondacks to sleep on hemlock boughs." Thus may it have been that the acorn thus planted in his early life has become the beneficient oak of the Trudeau Sanatorium.

What little need to refer to many conditions where accurate and

timely counter-irritation confers instant relief and markedly promotes cures. Cupping over a pleuritic stich, sinapism over the sacrosciatic foramen, blister over the area of the common duct will hasten the resolution of an acute cholecystitis and duodenitis, a resulting increase in the lumen of the common duct is sufficient to permit the egress of inspissated bile and catarrhal hypersecretion, and frequently quantities of small calculi. Indeed, I believe I have coaxed some fairly good sized ones to make the transit with the added aid of narcotic relaxation. Absorption of the sluggish fluid of a synovitis, pleuritis, or peri-carditis by blistering can often be better promoted than by attempts by more modern aspiration. Who has not observed the instant, at least temporary, change from a bronchial to a vesicular respiration by wet cupping over a congested and even an infiltrated lobe, and also reduction in the interpleuritic friction rales?

The obsolete leech will quickly despatch the pain and annoyance of an infiltration and inflamed hemorrhoid, and frequently secure results of a more pretentious operation. All such measures and many others conferring the same relief need no further mention. There is one, however, that I believe demands more serious consideration and frequent adoption, and that is venesection. This is but a form of counter-irritation in its direct derivative effect upon the general circulation as in cerebral congestion and some high arterial pressures, not due to an endoarteritis or calcification. Its supreme value is in a beginning pneumonia. I cannot conceive that any case however mild the character of the prevailing epidemic, whatever the age or the strength of the subject, can be harmed by a judicious blood letting. While we know the self-limited termination of ordinary cases, even in such cases where vigorous treatment has not been called for, there comes the time of tragic dramatic urgency as resolution approaches.

It is not of such expressions of the disease that I am speaking, but of those cases which develop at a time when previous ones in the vicinity have proved rapidly fatal. How common the expression, "Pneumonia is very fatal this season." Two such cases have occurred in my own experience recently. One died in twenty-four hours from the time I was called, the other in seventy-two hours; in neither of

these cases did we bleed and yet all the relief we seemed to afford was temporary and that by wet cupping.

I know of several cases of equal severity in our city during the past few months where every measure excepting venesection was most intelligently brought to bear. In private practice, time can be illy afforded to determine by laboratory analysis a classification that may justify an intuitive conception of rapidly impending toxæmia. I believe we delay while a right auricle is becoming dilated, and too cautious and fearful of taking too much blood, when a pound, perhaps two pounds or more, would not only relieve the auricle but rid the stream of bacilli and their toxins and the hyperinosis which must be their ally. Absence of a venous turgescence or an accompanying low arterial pressure should not prevent making the attempt to change the equation into one of more equality in the early defense of the attack. The average adult has from twelve to fifteen pounds of blood. Between the second and third day of pneumonia from two to three pounds is locked up in his lungs, or perhaps more in a short time, as the raucous sounds show lobe after lobe becoming successively involved. How much better, it seems to me, if this quantity could have been spilled into a basin in the early fight. Then better would come the opportunity to support the heart by well chosen and recognized agents that are known to be of undoubted value. Then the better chance to fight the toxæmia after it has been struck such a decided blow. May it not be true that such a force exerted against it will afford a more promising opportunity for supportive treatment by all other valuable means. Concerning this my mind is deeply engaged.

My caption and time will not permit more than a mere mention of an agent that I hope can be tried. This is alcohol, pushed, if need be, to a point of intoxicating narcosis or in such quantity and frequency of dose as the patient can be made to take and under constant observation of all occurring symptoms for a sufficient time to determine the immediate and contingent effects.

Before the days of antitoxin, malignant diphtheria was often successfully treated by almost unlimited doses of whiskey or brandy and while perhaps either of these may be preferable to proof alcohol, yet, it would seem likely that this suitably diluted with water would

more quickly exhibit its antitoxic power. The venom of reptiles is said to be neutralized by quantities of alcohol which would likely prove lethal in health. Whether this is true or not, it is fairly presumptive that it must be as dependable as anything in inhibiting the rapidly forming thrombi caused by the chemical constituents of the venom. Whether bacterial toxins produce thrombi in the thermal centers or elsewhere or upon whatever mechanism their destructive power consists, it is true that at the aortic orifice and arch is found an ante morten fibrinous thrombus in which toxæmia must have played its final rôle. Alcohol is known to be bactericidal in full strength and makes the transit of the circulation unaltered. Whatever may be the reduction of its antitoxic power in the quantity that can be made to mix with the rapidly infiltrating toxins, it must have some advantage if exhibited coincident with, or immediately following a substantial reduction in the toxic poison by adequate bleeding. There are theories pro and con of the action of alcohol upon the heart. Whatever of fact there may be in regard to this, it seems reasonable that a heart required to stand the effects of alcohol for a sufficient number of hours, to exert its hoped for antitoxic power, can well be depended upon to behave better than when required to withstand the onslaught of an unrestricted toxæmia.

If these meagre thoughts shall engage the attention of any one, and, if he shall find his mind receptive, I hope he may elaborate his ideas and in good time apply them. If I must meet again such awful cases as the two to which I have briefly referred, I shall use all possible persuasion with my consultants to gain their consent to a trial. We can at least have no greater depression than comes even when it is generously said by professional friends and relatives, "You have done everything possible, we must die when our times comes." Perchance the good clergyman will come with his benediction, "It's God's will." Then must come the answer to our clinging conscience: Did we keep God's "powder dry"?

AN APPRAISEMENT OF THE EXAMINATION OF THE HEART WITHOUT INSTRUMENTS OF PRECISION.

By FRANCIS J. DEVER, M.D.,

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There seems to be a growing tendency in the field of cardiology to regard the use of instruments of precision as of overshadowing importance. Very recently there appeared in one of our medical journals a paper in which it was stated that percussion was utterly untrustworthy and that no adequate conception of the size of the heart could be formed without the use of x-rays. We all are eager to testify to the great advancement in cardiology that has come from a study of the heart by means of the polygraph, electrocardiograph and other mechanical aids; but let us not fall into the serious error of emphasizing these aids at the expense of the training of our special senses. The latter we take with us to the bedside of each patient; the former are avialable in but a small minority of cases.

Indeed, so much information of clinical value is obtainable by a properly made physical examination of the heart that only the minority of cardiac cases require the use of instrumental methods for the determination of the nature of the affection. The examination, however, must be made in a systematic manner and the special senses used must be developed to a high degree. There is no better system of physical examination than that taught for so many years in all medical schools, namely, inspection, palpation, percussion, mensuration and auscultation. Dr. Judson Daland, in his lecture to students, very pertinentaly adds a sixth caption — cerebration. The more one reflects on this sixth caption, the more one is inclined to underscore heavily its importance.

Physical examination fails more often because of poor technique and insufficient development of the special senses of the examiner, and less often because of physical factors in the thorax of the patient. A careful examination of the heart cannot be made hurriedly. Sufficient time must be given to ascertain all the facts obtainable. This

is particularly true of inspection which too often consists in a casual glance at a partially uncovered thorax while reaching in one's pocket for a stethoscope.

Of great importance is the location of the apex beat. This is sometimes rather difficult but is possible in the large majority of cases if sufficient time is given. The position of the apical impulse often is of great aid in formulating an opinion as to the significance of a murmur heard in a heart examined for the first time. A patient with acute rheumatic fever with a history of previous attacks has an endocardial murmur. The question arises whether the murmur is the result of an acute endocarditis or the result of a previous infection. With an apex beat in the normal position, the probability is that the murmur is the result of a recent infection. Occasionally, one sees a patient in whom a murmur has been reported and who has been told, perhaps years before, that he has a "leaky heart." If on examination the apex is found to be in the normal position, even though a murmur be present, one can rule out, in the absence of other signs, actual disease of that heart.

Contrary to the opinion of some, the heart may be outlined quite accurately by percussion. To accomplish this, however, the percussion must be very light. Indeed, the note produced should be almost inaudible. It is probably accurate to state that the louder the percussion note the more inaccurate will be the outlining of the heart. The sense of resistance perceived in the percussed finger is the guide and not the note produced.

In connection with the location of the apex beat and the percussion of the area of heart dullness, mensuration is of great assistance and prevents to a large degree error of judgment of the actual size of the heart. In mensuration the anterior midline of the thorax is used as the fixed point, and the distance from that point to the apex beat is measured in a horizonal line. The average normal length of this line is 8 cm. in men and 7.5 cm. in women. The average normal total width of heart dullness is 9 to 10 cm. Mensuration obviates the possiblity of being mislead by the finding of an apex beat in the midclavicular line in a patient with a narrow thorax.

In chronic hypertensive nephritis with pulmonary edema, there arises the necessity of differentiating between toxemia and right heart

dilatation as the cause of the edema. If mensuration reveals an increase in the length of the horizontal line of heart dullness to the right the cause of the edema is, in all likelihood, cardia.

In auscultation the most important element is the study of the muscle tone of the heart. This tone must be considered in relation to the size of the heart. An hypertrophied heart that produces a tone no greater than that produced by a heart of normal size is a heart with weakened muscle. This is a point in physical diagnosis that is all too frequently neglected. And yet, a comprehension of the state of the myocardium, the prognosis and the treatment all revolve about this sign. Murmurs are of decidedly lesser importance than the quality of the first sound. Premature contractions considered alone may have very little significance. Occurring in association with a diminished muscle element of the cardiac first sound they may have the greatest significance. To illustrate: A gentleman over sixty years of age complained of attacks of severe pain of short duration in the epigastrium. Eructation of air relieved the pain which seemed to bear no relation to the taking of food. He had been assured by two very excellent physicians that the pain was of gastric origin. Examination of the heart, however, revealed a poor muscle tone with an occasional premature contraction, revealing the cardiac origin of the symptoms. Two months later the patient died in an attack of epigastric pain. Death from such cause is erroneously attributed to acute indigestion.

In conclusion we wish to emphasize that the appreciation of the great value of instruments of precision in the study of disease of the heart must not lead to an underestimation of the importance of a properly made physical examination.

The physical examination must be conducted in a systematic manner and sufficient time taken and effort made to obtain all signs possible.

DISCUSSION.

DR. DUNHAM: The study of the movements, size and shape of the heart is not as simple as one ordinarily thinks. I want to warn you against the belief that you can accurately determine the size of the heart by percussion. If you will day after day check out your physical findings by the fluoroscope and x-ray plate examinations you will soon learn your shortcomings. But the study of the heart dullness by percussion is most interesting and valuable. By this

method you can determine the movements of the heart dullness with change in the position of the patient. Thus if you will percuss out the left border of the heart with the patient in a standing position and then allow the patient to lie over on the left side and again determine the left border, you will get the marked movement of which the heart is capable. This can be very easily checked out by fluocoscopic examination.

In the use of the fluoroscope and x-ray plates let me state that the purpose of instruments of precision should be to prove our diagnosis, not to make them. When we get this fundamental idea firmly established in our minds it will be of the greatest value in our clinical studies.

Dr. Hall: I have been percussing the heart for forty years. It is a very dependable measure. I may point out that I make the diagnosis of a great many cases practically by percussion alone, as in a recent case of pulmonic stenosis, verified by the x-ray.

Dr. Claytor: I have done a great deal of work on heart percussion, in fact, the first paper I read before this society was upon this subject, but I have become convinced through orthodiagraphic studies that it is very difficult to be accurate as to the right border of the heart.

The left border of the heart and the width of the aorta, are not so difficult to outline. I have, however, learned to depend more upon bimanual palpation to detect a dilated aorta. According to Hoover of Cleveland, the sensation of pulsation elicited by this method of examination is conveyed by the muscle sense of the chest and shoulder muscles of the examiner rather than by the throbbing of the aorta against the fingers on the anterior chest wall.

While I cannot agree with Dr. Hall that the percussion of the heart can be done with such great accuracy, I believe that in most cases the experienced clinician should be able to detect a dilated aorta or heart without the aid of the x-ray.

Dr. Dever: I must accentuate that it is not the purpose to discontinue instruments of precision. The idea is not neglect the training of our own special senses. The subject of my paper was suggested by internes and postgraduate schools. Therefore, while I hesitate very strongly to see such an elementary topic before this Society, many of our men, being teachers of medicine, will go back to their schools to make these medical students do physical examination.

Dr. Robt. Wilson: I have been gratified to hear the emphasis that has been laid upon the importance of physical examination. Instruments of precision are, of course, indispensable in diagnosis, but I think we should teach our students to regard them as furnishing only confirmatory evidence in most cases.

REPAIR OF HEARTS THROUGH PERFORMANCE OF MOVEMENTS.

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First a personal episode and then an offering of experiences and interpretations in one direction.

In 1900 I read a paper before the American Climatological and Clinical Association—composed chiefly of experts in diseases of the heart and lungs—entitled: "Chronic Heart Disease in Children Relieved by Systematic Movements," which was published in American Medicine, May 1901.

During the discussion my recommendations were snowed under by adverse comments. One—that Master in Medicine and most lovable of men, Abram Jacobi—put a requiem upon my notions by asserting there was only one treatment for serious disease of the heart, and that was prolonged rest.

In those days there still lingered certain echoes of mediævalism, such as "crucify the vile body" and many traditional prejudices against wholesome motor functioning.

Among the notable advancements in knowledge none stands out more conspicuous and gratifying than that the body is a congeries of neuro-mechanisms and movements, whereby life is lived, health is saved, maintained and also regained, provided no destructive changes have become so serious as to render movements either unwise or to be wholly forbidden.

Tone, a property of muscle, is the prime index of health, and when present all the organs function economically. "The muscles are," as Michael Foster says, "the master tissues." Being "the great oxygen laboratories" they must function adequately otherwise dysfunction or malfunction is immediate and inevitable throughout. What is tone? The quality whereby a muscle is capable of full, prompt, and steady contraction, and also of equal importance of prompt and complete relaxation. This property of alternation of

maximum tension and of relaxation constitutes poise or balance between energies and structures. Abnormal over-action, either in prolonged contraction or in prolonged relaxation, shows disturbance of metabolism and of volition. Poise an essential condition of health and the starting point of all repair.

It is desirable to think in terms of tissue tone, of over or under-tone, in all clinical problems. This should aid much in acquiring a sense of clinical proportion.

Undue contraction is over-tone; undue relaxation is under-tone. During protraction of one or other stages or phases of action, wasteful leaks of energy occur.

We shall become more accomplished restorers of tone, of balance, of harmonious interacton, just so soon as we are guided in clinical classification by the natural or primal laws, and slough off the evil habits of elaborating endless minute subdivisions or differentiation. Medicine has only reached the stage where minutia and shades of phenomena are being diligently accumulated and categorized, as Sir James MacKenzie so constantly warns. They could readily and profitably be grouped, correlated and viewed in their due proportions and significances. As all roads lead to Rome: all symptoms point to central stations. We should aim to get back to the real starting point and strike outward on the most direct road to repair and waste less time groping from the periphery backward.

It may be distasteful to be forever harking back over the old trail we so nonchalantly have pursued in company with others whom we wish to overshadow by elaborate minutiæ. Always and ever that man reaches the goal first—or at least most certainly—who is most positive of his starting point and direction and objective. It is the tortoise and hare over again.

I often find myself forgetting or omitting some essential point in a simple technique which has often proved of value, especially when obstructions yield readily, the condition being relatively simple. When the state proves obdurate or complex and I concentrate; then practical steps come to mind and their use serves to turn the trick.

My contention is, and always has been, that for many, perhaps most, cardiopaths—especially children—the plan of depending upon protracted rest is inadequate; that a large, varied and important

group of benefits will ensue, as has now been amply demonstrated from carefully graded, guarded, guided natural movements. They may be done, should always be begun, while in the recumbent posture. As improvements grow in nutrition, oxygenation, in blood quality and distribution, all the organs tend to regain vigor.

In the paper referred to was related many gratifying personal experiences and many more have occurred since. The diagnoses in these earlier cases were by no means exact, yet no untoward effects occurred. Now, in the light of newer advances in cardiac structure (His, Sir Arthur Keith, Tawara, etc.) also in the explanation of cardiac irregularities—till then merely confusing conjectures (Sir James Mackenzie, Sir Henry Lewis, etc.)—anyone can avail himself of *experts* in the polygraph, electro-cardiograph, and then proceed from the exact knowledge supplied.

However, these elaborate and delicate instrumentalities demand great patience and exactitude in technical training, hence only experts can use them or interpret the findings, or link them up with problems of developmental peculiarity, of adequacy or inadequacy, or of progress in pathologic changes. Moreover these new sources of enlightenment seem to be largely preëmpted by the clinical laboratory researchers, also by the pharmacologic and manufacturing researchers and technicians. The chief value of the medical attendant, or personal adviser, is the degree and quality of his abilities to exercise common sense and judgment in replacing disarrangements of gear, adoplalem and in reëstablishing motor functioning and its performance.

One fact increasingly impresses me, viz.: the curative potency in most disorders, whatsoever be their origin, of regulating blood flow by rational agencies, such as by induced movements, by pressures, by releasing from compressures, by tractions, by leverages, also by contrasts of heat and cold, especially by insisting upon suitable voluntary movements. Unless motor performances are insisted on and guided, certain hampering effects must persist which lower the co-efficient of functional integrity.

Of course any or every other means may be also used but under no circumstances will, or can, the results be in accord with our

resources unless, or until, these also are employed, thereby constituting a totality of clinical effort.

The interest, or custom, or trend of the medical consciousness has as yet moved little in this direction; certainly not in proportion to the clinical needs and justifications.

The question is one primarily of bio-hydraulics, including competency in the reflex circuits, of adequate nutrition in the cell bodies of the cord—metameres—and integrity of the neuro-muscular mechanisms; also of the qualities of the blood as a tissue. Until these are supplied, how can we rely on medication to do its expected work, or the organism to furnish the right background or defense or repair adequacies?

My experience brings me clinical problems of individuals who are at what the late Dr. S. Weir Mitchell used to call "the dead point" of suspended or retarded progress. By locating the obstruction, by compensating or removing it, or supplying the needed release of energies, or of push-over, they go ahead satisfactorily.

Long ago I became impressed with the value of accurately directing the potency of voluntary movements and demonstrating them as being the one, or the chief, thing lacking in protracted heart disease in children. I am emboldened to believe some of my earlier methods were then better than those of some others, and hence again submit a sketch of my procedures. My contentions seem to have been proved by the now large number of special clinics—nearly fifty in New York City—and convalescent homes, such as the Burke Foundation at White Plains, New York.

These voluntary movements I at first administered (empirically) in the recumbent posture, by simple extensions, flexions, torsions, and tractions, in a way later to be described. The then almost hopeless cardiopaths steadily gained. A host of others have made equal and greater successes along the same lines.

To repeat: I appraised the conditions from the standpoint of local and general stagnation, from disuse of motor mechanisms, which demand use—exercise—and are of particular importance in the childish or growing organism. While in school and college I was a trainer of athletes. From these experiences certain principles emerged which as now submitted are in full accord with fundamental

or natural laws of muscular physiology. Since it is accepted that performance of function—especially motor function—makes structure, not only muscle structure but that of all the organism, hence the performance of movement remakes structure and thereby enhances function and repair.

Thus the constructive cycle proceeds to the benefit of not only the healthy but to the recovery of health.

It is merely a matter, or question, of how nearly rightly the process is initiated and carried on; in order to do no harm and yet do the most good where and when it is most needed.

My principles of reparative movement are these: starting each and every time from a position of relative advantage in which the part and also the remainder of the organism is at ease, e.g., the recumbent or reclining posture; the limb being held in the operator's hand and the particular movement determined being plainly indicated by stroking the muscles to be used. The form of the movement is always from zero—or full relaxation—with increasing force to maximum or the fullest tension. This rule (I call it my "law") obtains whether the movement be extension or flexion, or torsion. Always this order is preserved. Thus the power is put on gradually from nothing to most; held there for an appreciable time—say two seconds—counted.

Each movement should be directed from start to finish. Count one to five then hold two—total seven. Thus: go—one, two, three, four, five—hold one, two.

By indicating the movement I urge—as has been later described by Colin McKenzie (in "The Action of Muscles"—the best conditions of advantage are secured thus: hold the part with one hand and touch the muscle called on to perform with the other. Trace it from origin to insertion. The operator also himself makes the same movement with his own corresponding muscle and shows the action graphically.

Thus there is a plain visualization of the action indicated. The part to perform should be supported—as nearly as possible—in a favorable position to perform.

This is the method to pursue in case of a partial paralysis, whether the motor disability be due to *central* defect (as in polio-

myelitis or cerebral defect) or due to disuse compulsion—as after trauma or post-operative effects. So much for limbs. The same rules should obtain for the muscles of the neck; the lateral movements of the spinal column; also the abdominal and thoracic parieties; also the posterior abdominal—the psoas, the iliaci and quadratus lumborum.

Then the chest and trunk and pelvic girdles should be educated. Also of importance are the erector spinæ muscles, the backward bendings; resting on head and buttocks, later on head and heels.

I urge *special attention to the lateral movements of the spinal column.* By this means the great sympathetic trunks are alternately put one upon the stretch and the other fully relaxed. This—as Winsor and I have shown—is of powerful efficacy in stimulating the sympathetic functioning. (See articles by Taylor and Winsor on Spondylo-Pathology, American Medicine, August and November, 1922).

All this—be it observed—is so gently and gradually done that no harm can ensue. The operator can—and should—make frequent observations as to pulse rate and volume; also respiratory quickening; also color (countenance) reactions. Thus the patient can be lured on and on to greater effort and in absolute safety. He will gain in confidence and skill and precision; also in power of muscles and in particular of heart muscle, and all the arterial muscles.

The problem of maintaining or regaining full integrity of the heart and blood and circulation is one of preserving and enhancing the defense and repair mechanisms at their best. This includes adequacy in development to achieve a full endowment for resistence or existence; also by so fortifying the organism that it shall rise superior to perils of environment at all times and under all circumstances.

If, or when, the heart yields to some injury, disease or infection then the practical procedure becomes one—largely—of devising and applying means to prevent a harmless anatomic defect from developing into a dangerous disease; as for example: latent tuberculosis. Henry Sewall contributes a highly suggestive and practical paper entitled: "One Road to the Control of Heart Disease," (J. A. M. A., June 2, 1917) from which we may profitably take a point or two.

"Just as civilized mankind harbors almost constantly a more or less latent tuberculosis, so infective disease of the heart, provoking no classical symptoms, but potential for catastrophe, is well nigh invariable at some period of life. The practical conclusion from such a premise is that, as in the case of tuberculosis, the latent anatomic derangement may break down into functional inefficiency in an unfavorable environment, such as added infection or over-use."

"Only on the assumption of the universality of cardiac infection will the clinician give adequate consideration to morbid signs which point to cardiac disease."

We are concerned with those cases "in which there is a dangerous narrowing of what James Mackensie calls the field of physiologic response."

"It is doubtful if the student keeps clearly in mind that valve lesions are probably all due to infections conveyed not by the intracardiac flood which bathes the valves but by the capillary streams which spring from the coronary circuit, and the slightest attack on a valve presupposes a more or less widespread infection of the myocardium."

"A tissue which is the seat of a viable infection is like a seesaw: on one end is seated physiologic resistance, on the other infective virulence; the balance represents the ratio of the two forces. Precisely in the same way we frequently find that a quiescent pulmonary tuberculosis is lighted up into progressive disease by injudicious habits. Indeed, the salutary relation of rest in active tuberculosis is identical, with greatly increased emphasis, in acute heart disease. In tuberculosis, Nature is prone to forgive the careless transgressor; in heart disease, perhaps more rarely."

It has been my experience that sensory disturbances also yield to judicious curative movement. As Selian Neuhof says (Precordial Pains of Cardio-Vascular and Extracardiac Origin," J. A. M. A., March 4, 1916):

"Studies of the cardio-vascular nerve supply have shown that there are rich ganglionic and nerve plexuses which surround the heart and the root of the aorta and are formed by branches of the vagus and sympathetic. There are also ganglionic cells and nerve fibrils in the sino-auricular node, the auriculoventricular bundle and

throughout the cardiac musculature. So far as known nerves of sensation are absent. Through the fundamental work of Sherrington, Head and later Mackenzie, it is known that a viscus, though not possessed of nerves of sensibility, may, when irritated excite the corresponding visceral segment of the spinal cord; the latter then sends abnormal centrifugal impulses to the muscles, glands, etc., which in the skin give rise to morbid sensations usually felt and denoted as pains. In cardiac disease, the area ordinarily affected is the precordium; depending on the nerves involved, the intensity of the irritation, or possibly on the irradiation of the centripetal impulse to other spinal segments, the pain may spread to the entire chest, to both arms (especially the left), the fingers, neck, head, the interscapular region, the epigastrium, the abdomen and even the thighs. Besides the precordium and left shoulder the epigastrium is the favorite site for referred pains, a fact which often causes erroneous diagnosis, and mistakenly directs the therapy to the stomach."

How and why does it came about that the claimant for Physical Therapeutics is so little regarded in this forward going direction? His is a continuous performance of differentiation, of diagnosis and of prognosis, along with steps of treatment; of setting things in order by some modality of motion (Orthokinetics).

It is partly due—perhaps mainly due—to the absence of the voice of clinical authority whereby hesitancy, doubt, suspicion is abetted. Also it would seem that in most lines of experimental endeavor—outside of commercial and industrial lines, certainly within medical endeavor—what is strange is also to be denied, shunned, scorned. Until an idea gets by the barriers of ancient taboo, it is further branded as the product of visionary zealots or conscienceless opportunities, at best unworthy of attention.

And yet the heart and circulation, the reflexes of vaso-motion and viscero-motion are peculiarly amenable to regulation through eliciting and conditioning mechanistically the proper reflex responses. By balancing the reflex circuits, through judicious impacts at or near the spinal centers, it is well established that poise in the hydraulic and other mechanisms can be achieved.

Furthermore such straightforward influences from centers to periphery possess a merit all their own, and superior to those by

drugs introduced into the body, where action can then be only partially readjusted. They are safer, surer, more readily adapted to the needs of locality and time; they can be cautiously applied or reversed, step by step as the needs of the situation become revealed.

The effects are more controllable since they can be—like those of an insentient engine—reversible on the instant, changed in direction and degree, in short a true passing from plane to plane, gradient to gradient, as seems wise.

SYPHILITIC AORTITIS.

By THOMAS A. CLAYTOR, M.D.,
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From statistics, which I will not give in detail, it would appear conservative to say, that more than fifty per cent of the syphilitic show involvement of the aorta, and Lentz, (Med. Klinic, 1913, p. 955) states that in large cities 20% of all syphilitics die of aortitis, as against three or four per cent from paresis, one or two per cent from tabes, and ten per cent from all other syphilitic affections, as of brain, liver, kidney, etc.

Pathology.

The researches of Heller and his pupils, and Chiari, (since abundantly confirmed in other laboratories), have proven the primary lesion to be a small celled, or granulomatous infiltration of the media along the course of the individual vasa vasorum. (Adami & Nicholls, Principles of Pathology, 2nd Ed.) Accompanying the infiltration, there is a well marked atrophy of the muscular and elastic elements of the medial coat. The absorbtion of the elastic tissue is especially striking (Klotz). The process does not extend primarily into the intima, but secondarily this coat unergoes a proliferative thickening. At a later date, when fibrosis has given way to atheroma, the vessels extend into the necrotic area. In other words, a syphilitic mesarteritis is followed by intimal sclerosis and its sequella.

Portion of the Aorta Involved.

While any part of the aorta may be the seat of syphilitic disease, that above the diaphragm is most often involved. According to Hoover, (Jour. Am. Med. Assn., Jan. 24, 1920), the ascending, or first portion of the arch, shares in the syphilitic process in 95 per cent of the cases, and in the majority of the cases, the pathologic changes are confined to this portion of the arch.

Klotz, (Am. Jour. of the Medical Sciences, Jan., 1918) suggests that this selective preference is due to the especially abundant lymphatic supply to the ascending and transverse portions of the aorta. The disease begins just above the aortic valves and spreads upwards toward the arch or downward to involve the tissues about the aortic opening, either the ring or the valves or both. Instances have been reported where the ring was so affected that there was a relative aortic insufficiency, with the valves themselves in perfect condition. The coronaries may be involved directly or their aortic openings obstructed by plaques in the aorta.

Period at Which the Aorta Becomes Involved.

From the experimental work upon rabbits conducted by W. H. Brown and L. Pearce, (Archives of Dermatol & Syphilol, Chicago, Oct. 1920, 2, No. 4, p. 470) it was demonstrated that immediately an infection takes place, the spirochetes begin to multiply and invade the surrounding tissues, gaining access to both the lymphatics and the blood stream, and are widely distributed over the body even before an initial lesion can be detected. If this be the case, the aortic involvement might begin at once.

As instances of early involvement of the aorta, may be citied two cases of Harlow Brooks (Med. Record, N. Y., 1912, LXXXI, p. 351), in one of which there was serious aortic disease before the secondary rash had appeared, and in the other, within six months of the primary infection.

What might be called the period of latency is variable, and may be put down as from a few months to more than fifty years, however, a fair average would be about fifteen years between the primary infection, and the appearance of symptoms indicating an involvement of the aorta. This then would bring the majority of these sufferers to us between the ages of thirty-five and fifty years; much earlier than is the case with atheroma.

Clinical Divisions.

Clinically, cases of syphilitic aortitis may be divided into three classes.

First. Those in which physical signs are not demonstrable, or are so slight as only to suggest aortic disease.

Second. Those in the intermediate class who, because of more advanced disease, show definite signs of dilatation of the aorta, or a lesion of the aortic valves or ring.

Third. Those far advanced cases of aneurism or aortic valvular disease with decompensation and all its well known accompaniments with which we are so familiar in the wards of our general hospitals.

Those of the first class, which would, of course, offer the most hopeful field for cure are, for obvious reasons, seldom recognized.

Those of the third class have, in my experience, seldom shown any benefit from anti-syphilitic treatment, so that our chief interest is centered upon the second class.

How long disease of the aorta may exist before symptoms appear, of course is uncertain, but with extension of the process, there may result the following anatomical changes with their clinical manifestations; diffuse dilation of the arch, aneurism, aortic insufficiency, obstruction of the coronaries.

Symptoms.

The most important symptoms are pain, dyspnea, cardiac irregularity, hoarseness and fever. Pain is the most constant symptom. The location is substernal usually, though it may be felt to the left of the sternum, and even toward the apex of the heart. The intensity varies as does the radiation. It is sometimes spoken of as more of a feeling of pressure, and again as an agonizing pain identical with that of a true angina, radiating to the shoulders and down the arms.

Diagnosis.

As is the case in so many other diseases, the most important element in favor of making a correct diagnosis, is to be on the watch for it, remembering that: "*Syphilitic aortitis is responsible as shown by autopsy statistics and the Wassermann reaction, for most aneurisms, about seventy-five per cent of the cases of aortic insufficiency in adults, many cases of dilatation of the aorta, and a certain group of cases of angina pectoris.*" (Longscope) (Arch. of Int. Med., Jan. 1913).

With the symptoms already mentioned, the condition should at least be suspected in the presence of an aortic murmur either systolic or diastolic or both; a dilated aorta, which can be recognized in most cases by a careful physical examination, and corroborated, if necessary, by the x-ray, the history of an infection; the presence of other syphilitic manifestations,—such as the loss of knee jerks, unequal pupils, etc., or as I have happened to have seen in one or two instances, an external gumma. And last but of the greatest importance, the Wassermann reaction must be our most reliable aid, for many times a syphilitic infection is denied, whether wilfully or through ignorance it matters but little.

Treatment.

As syphilitic aortitis has a destructive pathological process consisting, as has been said, of a round celled or granulomatous infiltration about the vasa vasorum, destroying as it does, the muscular and elastic fibres of the median coat primarily, thereby causing a local or more or less general weakening of the aortic wall, the more promptly specific treatment is instituted, the better will be the chance of cure.

While it is most unlikely that any remedies can cause a regeneration of muscular and elastic tissue, there seems no reason why the destructive process cannot be arrested in many cases so that nature's method of repair by the formation of fibrous tissue may go on.

An injured valve remains injured, and a dilated aorta remains dilated, but the ravages of the spirochetes or the result of their presence in the tissues may be arrested.

Active treatment if instituted early enough, will many times give very gratifying results. The pain which is a most constant symptom is often entirely relieved, and the dispnea greatly lesened, and the patient be restored to comparatively good health, though of course, eternal vigilance, to prevent a recrudescence, is necessary.

A six to eight weeks course of salvarsan, followed by mercury salicylate for the same length of time has been found satisfactory. This, of course, is but a preliminary and may have to be repeated a number of times at varying intervals of months or years.

The same difficulty is met in these cases of aortitis as in other manifestations of syphilitis in convincing sufferers that the cessation of symptoms alone, is no positive evidence that the disease has been eradicated.

Attempts to relieve anginal pains by the use of antispecific treatment in cases where there is no positive luetic history, and a negative Wassermann will be generally unsatisfactory though, of course, there is always the chance that a syphilitic aortitis may be the underlying cause.

In Conclusion.

1. Syphilitic aortitis is a distinct entity.
2. It is not uncommon.
3. It should be recognized much earlier than is the rule.
4. It produces distressing and serious symptoms.
5. Great relief of symptoms and probable arrest of the disease often results from proper treatment.
6. Treatment should be active and persistent.

THE ROLE OF FORCED VIBRATIONS IN THE PRODUCTION OF PHYSICAL SIGNS.

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This paper presents in outline a few of the principles concerned in forced vibrations and in the application of these principles to the explanation of some of the physical signs encountered in pulmonary and pleural conditions.

In order to avoid unduly lengthening the article very little is said about the evidence on which many contained statements are based, that is, evidence derived first, from prominent physicists, secondly from the literature, thirdly, from animal experimentation, and fourthly, from clinical data.

In the development of auscultatory phenomena between their place of initiation in the body and their reception by the stethoscope at the chest surface, three outstanding factors are recognized, namely, diffusion, reflection, and forced vibration.

The first two of these are simpler, more generally understood, and more easily traceable in many instances than is the third.

Diffusion applies to that loss in intensity (energy) of sound waves due to dilution or dispersion consequent upon their spreading out through a progressively increasing space. In a medium in which the absorption is small enough to be neglected, and which is extended enough, (infinite), so that the complications introduced by its boundaries may be disregarded, the intensity of a sound varies inversely as the square of its distance from the source. Typical examples are, the reduction in intensity of the vocal resonance in suitable cases of pleural effusion, and the normal progressive loss in intensity of the vocal resonance as one moves the stethoscope downwards from the lung over the liver.

The term reflection implies a forcing back of sound waves, that is, an obstruction with consequent diminution in intensity of these

waves in their regular forward progress. It is apt to be associated with a certain degree of absorption of sound, which then becomes transformed into some sort of energy. The phenomenon is frequently met with in the chest where sound waves encounter changes in media of different density, for example, air and tissue or air and fluid. Examples are found in cases of pneumothorax with diminished vocal resonance, and in the normal lung, where the weakness or absence of the whispered sounds depends largely on reflection.

The same factor of reflection may also favor the transmission of sound waves under certain conditions, for example, through the speaking-tube effect of the trachea and bronchi, which carry waves deeply into the lung with a very considerable preservation of their energy by preventing diffusion.

Both diffusion and reflection, as here used, affect all sounds alike, whatever their pitch, as far as demonstrable evidence is available in the case of the chest, except in those cases in which a reflecting body instead of reflecting is set into forced vibration by encountering other vibrations of its own frequency. Diffusion and reflection, moreover, do not involve, as an integral part of their operation, the development or association of new sources of sounds.

Forced vibrations, on the other hand, are concerned especially with vibrations of limited ranges of pitch, and involve the development of new sources of sound, secondary to the primary or initiating source of the sound.

To clarify the subject at this point it may be well to mention three elements concerned in physical sound. The first is the sonorous or sound producing body, for example a tuning fork or the vocal cords set for speech. Secondly we have the effects of such a vibrating sonorous body, which may be revealed as sound waves or as the setting into vibration of a new sonorous or sound producing body, as exemplified by placing a vibrating tuning fork in contact with a suitable piece of wood. There is also a third element, namely, the force that sets a sonorous body into vibration, for example, the tapping of a tuning fork, the expiratory passage of air over the vocal cords in speech, or vibrations of a suitable pitch that excite forced vibrations.

Definition of forced vibrations. When a sonorous or sound

producing body, possessing a certain period or periods of vibration peculiar to itself, is set into vibration by external vibrations having its own, or somewhere near its own, natural period (periods) of vibration or frequency, it is said to be forced into vibration by these external vibrations, that is, is set into forced vibration, and, under suitable circumstances, gives rise to a new sound. A sonorous body (like a suitable piece of wood) may be set into forced vibration first, by direct contact with another sonorous body of proper frequency of vibration (pitch), as when a table is placed in contact with a vibrating tuning fork, or when the wood of a stringed instrument reinforces the string, in both these cases the wood becoming an additional source of sound. Secondly, a sonorous body may be set into forced vibration without any such direct contact with another sonorous body but in response to sound waves of proper frequency, for example, when one tuning fork is set into vibration by another vibrating fork of similar frequency but separated from it for some distance by air, in which case the sound waves in the air are the direct or immediately exciting cause of the vibration of the second fork, which itself then adds a new sound to that of the first fork.

A sonorous body then may give rise to sound in response to various exciting factors, for example as commonly met with in percussing the chest, or as a result of encountering rhythmic vibrations of suitable frequency and sufficient energy.

Some sonorous bodies are set into vibration by other vibrations of only a very limited period or frequency of vibration. Thus one tuning fork will respond to another of the same pitch, but ordinarily not at all to one of slightly different pitch. Other sonorous bodies like the wood of a violin, respond to vibrations of many pitches coming from the strings, but even this wide capacity has its limitations.

The term "forced vbration" is herein employed to include a number of terms that are used more or less vaguely and with different meanings, for example, resonance and sympathetic vibrations.

It is important in order to prevent confusion to clearly differentiate between forced vibrations and simple sound waves, the former being capable of producing the latter, while the latter simply

transmit the energy originated by sonorous bodies, that is, "conduct" the sound energy.

The rôle of forced vibrations in the explanation of physical signs. Inquiry into this subject naturally follows two lines. One is concerned with an analysis of sounds at their origin, especially as to their pitch and intensity. The other is concerned with the various structures or combinations of structures and materials between the original source of sound and the chest periphery, and seeks to determine to what extent these structures can be set into forced vibration, and what is their natural period or frequency of vibration. Assuming that a sound of sufficient energy posseses a given pitch at its origin, and vibrations from this original source encounter media or structures of the same natural period of vibration, we would look under proper conditions for an amplification of sound from the new sonorous body that has responded with forced vibrations.

Analysis of sounds at their origin. An analysis of many of the sounds we are at present chiefly concerned in, namely, those originating in the upper respiratory tract during breathing, whispering and speaking (or singing), presents great and unfortunate difficulties, and for many of these sounds is in a very incomplete state of development. Physicists have been concerned chiefly with the analysis of musical and so-called rhythmical sounds.

It appears agreed by physicists today that all audible sounds are made up of one or more pure tones, that is, of individual sounds having a single vibration period, as exemplified in the case of the tuning fork. No such single sound is produced in the upper respiratory tract. The simplest form of sounds comes from the vocal cords during speech, but even these are composed of both fundamentals and overtones. All other sounds under consideration produced in the upper respiratory tract are much more complicated, consisting, presumably, of many individual sounds varying in pitch, intensity and duration. These complicated whole sounds still await anything like complete analysis. They are produced in the upper respiratory tract during breathing and whispering. They also include all the sounds of speech except those produced by the vocal cords. In other words, the sounds made during speech by the lips, teeth, etc., and by the air escaping during expiration, fall into this very complex class.

Of prime importance is it to know the energy or intesity of the elemental sounds making up any complex sound. In the case of the vocal cords it is known that the original vibration energy is quite weak, but that this original energy is greatly added to (amplified) as a result of forced vibrations (resonance) set up in the chambers of the upper respiratory tract. In the case of the vocal cords both the fundamental and overtone vibrations become greatly reinforced during speech. In the case of the other (non-vocal cord) sounds produced during speech, and in the case of the sounds produced in the upper respiratory tract during breathing and whispering, we have some idea of what the total volume of sound is in each case, but we do not know the intensity of the individual tones composing each of these complex sounds. It is likely that these individual tones are for the most part quite weak. This opinion is based partly on their multiplicity, revealed to some extend by the effect of forced vibrations on the whole sound. An individual tone thus represents only a small fraction of the total volume of the sound as heard. It is also based partly on the fact that none of these sounds yields clinically any great degree of intensity as a result of forced vibrations, such as occurs for example with vocal cord vibrations. The whole sounds produced in the upper respiratory tract during breathing and whispering become distinctly modified in other respects (quality and pitch) than intensity, for example, by the mouth and nasopharynx and also by bronchi and pathological cavities, but the total volume of sound rarely if ever becomes much amplified, indicating that whatever forced vibrations have been excited have been affected by tones of feeble intensity, these feeble tones alone having only a fraction of the energy of the sum total of all the tones making up the original sound.

Our conception, then, of all these sounds however loud, except those produced by the vocal cords, is that they are made up of individual tones that are much feebler than the whole sound. Such feeble sounds when amplified, whether 10 or 100 times, may still be relatively weak, and not add much to the original complex sound volume though distinctly affecting its quality. Only the vocal cord vibrations reveal much amplification in the upper respiratory tract.

In the case of the bronchial tubes the natural vibration period

of the contained air is of considerable frequency (high pitch) as far as clinically revealed, for example over solid lung.

With suitable cavities communicating with bronchi the natural period of vibration varies in frequency, probably usually being greater than that of the normal lung or that of the fundamental vocal cord vibrations in the male.

The natural vibration period of a pneumothorax cavity is variable, apparently sometimes more frequent, rarely if ever much less frequent than that of normal lung tissue in a normal state of distention. The coin test gives a typical example of a resonance (forced vibration) phenomenon, the pneumothorax cavity apparently amplifying certain tones composing the sound produced by the coins.

The resonating chambers represented by bronchi and cavities do not ordinarily yield any great increase in sounds produced in the upper respiratory tract during breathing, whispering or speaking. All three classes of sounds receive some modification yielding a change in quality, the resonating chambers being set into forced vibration by single tones, or combinations of a limited number of tones of closely corresponding frequency, these single or associated tones forming a small part of the whole sound coming from the upper respiratory tract. Thus the breathing and whispering sounds over the trachea may become higher in pitch over a solid lung due to resonance (forced vibration) within the bronchi.

The normal lung gives us a particularly clear demonstration of the contribution of forced vibrations to physical signs, especially as revealed in normal vocal resonance. The natural vibration period of the normal lung is relatively slow (low pitch), and is revealed to a variable extent on percussing over normal chests, some chests yielding a more accurate determination than others. The pitch of this note roughly corresponds to that of the normal vocal resonance and of normal vesicular inspiration.

Sounds failing because of unsuitable pitch to set the normal lung into forced vibration are actually obstructed by the normal lung in their attempted passage through it. This is largely because the tense membranes forming the alveolar walls interfere through reflection with the progress of the sound waves towards the chest surface.

The normal vocal resonance consists largely or wholly of sounds

resulting from vibrations capable of setting into forced vibration the pulmonary tissues. Vibrations, to activate the lungs in this way, must possess sufficient energy and appropriate pitch. They are commonly found in the fundamental vocal cord vibrations amplified by the resonating chambers above the vocal cords.

The average man's voice yields good vocal resonance but if it is sufficiently raised in pitch without change in intensity the vocal resonance can be made much weaker and like that in many women.

Other vibrations (pure tones) coming from the upper respiratory tract during speaking, and those produced during breathing and whispering, are believed not to come through to the chest surface unless in very feeble form, the reason apparently being that such vibrations are either of unsuitable pitch (too high) to give rise to forced vibration in the normal lung, or possess too little energy.

These same fundamental vocal cord vibrations appear also capable of setting into forced vibration the chest wall, probably chiefly the muscular tissue. Suitable percussion of the chest over the normal lung readily yields a sound which shows that it is a sonorous body and inferentially capable of yielding forced vibrations. This percussion note is often of a pitch close to that of the lung frequency and of the fundamental cord vibrations. Clinically it is a striking fact that oftentimes deep pressure of the bell of a stethoscope yields much feebler vocal resonance than light pressure. The explanation appears to lie in the fact that firm pressure on the muscular tissue interferes with its capacity for being set into forced vibration. This muscular tissue differs from normal lung in that it does not interfere with the simple conduction of sound waves.

The normal vocal resonance then is believed to arise in the vibrations of the vocal cords, chiefly the fundamental vibrations, these original vibrations setting into forced vibration, first the resonating chambers above the vocal cords, this combined sound then setting into forced vibration the pulmonary tissue, and finally the muscular tissue of the chest wall also being set into forced vibration. Thus three new sources of sound may be set into vibration in the production of normal vocal resonance. Vibrations of a sufficiently different pitch not only fail to excite these new sources of sound, but

are actually impeded by the lung in their progress towards the surface.

It is believed also that these vocal cord vibrations are the only ones arising in the upper respiratory tract that yield audible sounds of any intensity over most of the normal chest under ordinary conditions.

The normal lung is not limited to a single period of vibration, though restricted within fairly narrow bounds. A person with a low pitched voice and good vocal resonance can distinctly increase the rate of the fundamental cord vibrations and still furnish a marked degree of vocal resonance.

The vibrations felt and heard over the chest during laughing and coughing are also traceable to vocal cord vibrations.

Abnormal fibrous tissue is believed also to be capable of producing sound under proper conditions, especially when it is both sufficiently attenuated and properly associated with air.

The acutely solidified lung presents a marked contrast in many physical manifestations to normal lung, notably in its acoustic properties. Such a lung no longer has the capacity of yielding forced vibrations, at least to any great extent. But instead of producing a new sound in response to suitable vibrations of a limited range of pitch of its own or nearly its own frequency, it has now become a relatively homogeneous medium as far as sound waves are concerned, and transmits waves of different pitch equally well, with little loss of energy except that due to diffusion.

Over the solid lung we probably obtain sounds much the same as those within the bronchi connected with the solid lung, save for a change in intensity, and some change in quality due to the disappearance of the weaker vibrations. In other words, the sounds present in the upper respiratory tract during breathing, whispering and speaking appear in the bronchi somewhat altered in pitch and quality, largely due to forced vibrations set up in the bronchi, and then pass to the surface of the solid lung altered chiefly in intensity.

Summary.

It is believed that diffusion, reflection, and forced vibration are the main factors in developing sounds between their origin in the upper respiratory tract and their arrival at the chest surface.

Forced vibrations are set up in a sonorous (sound-producing) body in response to the influence of vibrations of the same or somewhere near the same frequency.

A body set into forced vibration does more than simply conduct sound waves; it actually adds a new sound and thereby amplifies the original sound.

In order that external vibrations may set a sonorous body into forced vibration they must posses sufficient energy.

The sounds present in the upper respiratory tract during breathing, whispering and speaking are all complex or composite sounds, consisting of individual pure tones. The respiratory and whispered sounds present in the upper respiratory tract, also the sounds produced during speech, except those coming from the vocal cords, are probably composed of very many individual tones, varying in intensity but probably each one much weaker than the whole sound of which it is a part, varying also in pitch and duration. The pure tones coming from the vocal cords are fewer and after amplification in the upper respiratory tract may have a relatively large amount of energy.

It is believed that the fundamental vocal cord vibrations, at least in the average adult male, are considerably lower in frequency, (pitch), than all other, or most other individual pure tones that go to make up the sounds present in the upper respiratory tract during breathing, whispering and speaking.

The development of the normal vocal resonance exemplifies well the effect of forced vibrations on physical signs. Initiated by the vocal cord vibrations, chiefly the fundamental ones, the primary sound vibrations become amplified through resonance (forced vibrations) in the upper respiratory tract, and pass down the bronchi into the pulmonary tissue which is also set into forced vibration, and finally may set into vibration the muscular tissue of the chest wall. Other individual pure tones arising during speech, also the tones in the upper respiratory tract produced during breathing and whispering, are not sufficiently amplified anywhere to add much to the whole sound because of deficient energy and unsuitable pitch and, on reaching the normal lung, are actually obstructed in their passage and do not reach its surface or only in much attenuated degree.

The solid lung changes the picture. It has lost the forced vibration capacity of the normal lung and also its obstructive properties, becoming a good conductor for all sounds alike, so that the whisper, breathing and vocal resonance all come through and are much like those within the bronchi except for loss in intensity. Vocal resonance (and particularly vocal fremitus) over solid lung (and over the back of the neck) may be actually less than over the normal lung, because forced vibrations are not possible in the acutely solid lung and non-sonorous neck. At the same time the whisper comes clearly through the solid lung and back of the neck because of the good conducting properties of both for all sounds. The whisper, however, fails to pass through the normal lung, being obstructed (largely through reflection) by it, and being unable to set it into forced vibration by reason of its to high pitch, as compared to that of the lung.

Individual tones going to make up the sounds produced in the upper respiratory tract (barring vocal cord sounds) are probably relatively weak compared to the whole sounds of which they are a part, and this is one reason why their intensity is not greatly appreciated at the chest surface, sufficient energy being lacking to set up forced vibrations. They do, however, set up forced vibrations of limited intensity in the bronchi and cavities, etc., sufficient to change the quality of the sounds audible at the chest surface, even if total intensity is not much affected.

Fibrous and other kinds of tissue in the lung, and also air, are capable of being set into forced vibration under conditions not mentioned.

ALTITUDE SICKNESS THE RESULT OF HEMOGLOBIN DEFICIENCY.

By JUDSON DALAND, M.D.,

PHILADELPHIA, PENNSYLVANIA.

On July 6th, 1899, while in Philadelphia, at an elevation of about 100 feet, my red cells were 87% by the hematokrit, which is about equal to 4,350,000 per c.m., and the hemoglobin was 70%. Eight days later, while at Colorado Springs, at an elevation of about 6,000 feet, the red cells increased 5,042,424, or 10%. Two days later, while at Pike's Peak, at an altitude of 14,107 feet, the pulse was 73, respiration 24, both easily accelerated by slight exertion, and both quickly returning to normal after a few moments rest. As I remained only four hours on Pike's Peak, there was no important change in the hemoglobin or red cells, but the following day, while at Colorado Springs, there were 477,476 more erythrocytes. A slight occipital headache developed, which gradually increased and became intense, and upon descending, the pain gradually diminished and disappared shortly after arriving at Colorado Springs.*

On March 22nd, 1920, my erythrocytes in Philadelphia were 4,810,000 or 96% (Thoma Zeiss), hemoglobin (Fleischl) 92%, and after two months' residence in Colorado, at an elevation of about 7,000 feet, the red cells increased to 5,670,000, or 113%, and the hemoglobin 98%, an increase of 860,000 erythrocytes, and 6% of hemoglobin.

After living three months in Wyoming, in 1921, at an altitude of 6,000 feet, my red cells were 5,840,000 or 116%, and hemoglobin 99%, showing a slight increase of red cells as compared with 1920. In less than two months after arriving in Philadelphia, the red cells decreased 610,000, and five months later, an additional 260,000 red cells disappeared.

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*Reported at the meeting of the Rocky Mountain Interstate Medical Society, Salt Lake City, in July, 1899.

After a residence of three months in Wyoming, in 1922, at an altitude of 6,000 feet, the red cells were 5,710,000, hemoglobin 100%, or an increase of 740,00 red cells as compared with the blood examination made prior to leaving Philadelphia for Wyoming.

These observations clearly show than at an altitude of 6,000 feet, after a residence of two months or less, one may expect a physiological increase of three-fourths to one million red cells, as compared with sea level, and that this increase will gradually disapear at sea level, in about the same length of time.

In ascending Pike's Peak, I was accompanied by Dr. S. Edwin Solly, who was underweight, slightly anemic, heart moderately hypertrophied, secondary to mitral valvulitis, and a lung damaged by a previous tuberculosis. The only signs he developed were a marked increase in the rate of the pulse and respiration, after slight exertion, quickly returning to normal after a few moments of rest. Slight vertigo was induced by bending the body so that the head was low.

In 1900,* Dr. Solly observed twenty-five students at Colorado Springs, at an altitude of 6,000 feet, whose average age was 22, during an average residence of eight years, the average number of erythrocytes were 5,900,000, and the average percentage of hemoglobin was 101.7. H also observed that a short residence at this altitude produced a greater increase of erythrocytes and hemoglobin than a prolonged residence.

About two years ago, Captain R. G. Hingston‡ pointed out that at sea level the atmospheric pressure on the surface of the body per square inch was fifteen pounds. Assuming that the surface area of the body is 2,000 square inches, then the total weight carried by the body at sea level would be thirteen and one-half tons, whereas at 10,000 feet, it is nine and one-half tons, and at 15,00 feet it is seven and one-half tons.

At sea level, the air contains 20.96% of oxygen, and about one-fifth of the air breathed is pure oxygen. At 7,000 feet, the oxygen in the air is reduced 25%, at 18,500 feet it would be reduced 66%, or

*Transactions of the American Climatological and Clinical Association, 1891.
‡Indian Journal of Medical Research, April, 1921.

the equivalent of *one-third* of the amount of oxygen in a given volume of air at *sea level.*

The average number of red cells at sea level is between four and one-half and five millions; at 8,000 feet—six millions; at 13,000 feet —seven and one-half millions, and at 18,000 feet about nine millions.

This *increase* in the number of red cells closely follows the *decrease* in the percentage of oxygen in the air. The physiologic daily increase of red cells in high altitudes may be about 250,000.

These observations make clearly evident the reasons why a certain amount of time is required in order to adjust the amount of hemoglobin to the demands of the body for oxygen at varying altitudes, and why symptoms occur when an individual *quickly* moves from sea level to high altitudes.

Captain Hingston observed, while in the Pamir Valley, in the Western Himalaya, that it required about one week to become acclimatized to an elevation of 13,500 feet, and at this elevation, the erythrocytes in a Ghurka were 7,140,000, in an Englishman 7,402,000, and in a Kashmiri 7,700,000, suggesting that the Kashmiri are better suited for high altitudes.

The Kirghiz and the Sirakolis, two Nomad tribes, living permanently on the Pamir Plateau, altitude 13,00 feet, differ in no particular from the people of the Plains, excepting that they average 7,596,000 red cells per c.m. Other authors observe at this high altitude, physiologic cardiac hypertrophy and emphysema. As the natives from the Russian side average 7,920,000, and a European averages but 7,402,000, it is probable that the former by possessing 518,000 more erythrocytes, are better adapted for clmbing Mount Everest.

On ascending from 13,000 to 18,000 feet, the erythrocytes were 8,320,000 after a two days residence, and as complete acclimatization would probably have taken place within ten days, the number of red cells would probably have been 9,000,000 or more. Failure to become acclimatized to high altitudes is chiefly due to failure to produce sufficient hemoglobin.

In descending from a high to a lower altitude, the number of red cells *decrease* at the same rate as they *increase* during the ascent, and require about the same length of time, and this change

occurs, whether the individual be a visitor or a permanent resident of high altitudes. A native Nomad's erythrocytes numbered 7,595,000 at 13,000 feet, and was 5,060,000 after descending 5,500 feet, and the same change occurred in a European.

Experienced mountain climbers may feel nothing more than a slight fatigue at 22,230 feet, whereas others may show the usual signs of altitude sickness at 7,000 feet.

As hemoglobin carries oxygen and the red cells carry hemoglobin, both are important, but the amount of hemoglobin is more important than the number of the erythrocytes. In chlorosis, the red cells usually contain *less,* and in pernicious anemia, *more* hemoglobin than normal. The number of red cells should be expressed in percentage, so that by comparson with the percentage of hemoglobin, the amount of hemoglobin in each erythrocytes may be estimated.

The reason why the Duke of Abruzzi ascended the Karakoran, 24,600 feet, *without symptoms,* was probably because he made the climb *slowly.* It is probable that certain individuals can manufacture hemoglobin in sufficient amounts to become acclimatized to Mount Everest, 29,002 feet.

The average pulse rate at 13,000 feet was 85, but moderate exertion produced tachycardia and rapid breathing. It is obvious that *permanent* compensation for altitude does *not* take place through the heart and lungs, but *only* through an increase in hemoglobin. Occasionally, Cheyne-Stokes' breathing occurs, with periods of apnea of fifteen seconds.

Altitude sickness occurs in cattle, hogs, sheep and dogs, and when acclimatized, there is a corresponding increase in hemoglobin. Altitude sickness may be induced or aggravated by stormy or wet weather.

Temporarily, in an emergency, the body meets the *increased* demand for oxygen by *increasing* the rapidity of the circulation of the blood through the lungs, and by *increasing* the rapidity and depth of respiration, and we therefore can understand why patients with diseases of the lungs or heart must move *slowly* from low to high altitudes, and require *more time* to become acclimatized than a normal individual. We can now understand why a patient with

serious pleuro-pulmonary or cardiovascular diseases may become worse or die when he *quickly* moves from low to high altitudes.

Remarks.

1. The symptoms and signs induced by going from sea level to high altitudes, is fundamentally due to insufficient hemoglobin, and nature automatically preserves a nice balance between the amount of hemoglobin and the body's need for oxygen.

2. The increase in red cells and hemoglobin in high altitudes is purely compensatory.

3. Acclimatization for high altitudes is prolonged or made impossible by any interference with the function of the cells that make hemoglobin and erythrocytes.

4. The stimulating influence of altitude upon the narrow cells producing hemoglobin and erythrocytes is beneficial to convalescents from most diseases including those suffering from the systemic manifestations of chronic focal infection, provided the focus or foci have been removed, to ill health due to general causes such as overwork, for example, and also to simple anemia.

5. Growth is greatly stimulated, especially in the young.

6. Selected cases of cardiovascular or pleuro-pulmonary disease may be *benefitted* by *slowly* moving from a low to a high altitude, but *death* may occur if the change is made *quickly* in serious cases.

POLYCYTHEMIA VERA.

By DELANCEY ROCHESTER, M.D.,
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In 1892 Vaquez of Paris rescribed a condition, with report of two cases, of an abnormally high red cell count 9,000,000 or more and a high hemoglobin 130%, an enlarged spleen and a red flushed face. To this condition he gave the name of polycythemia rubra.

In 1903 Osler gathered together several other cases from the literature and reported some more himself and described the condition in considerable detail discussing the etiology not very satisfactorily.

In his description he says, "The three cardinal feautres are a change in the appearance of the patient, enlargement of the spleen, and hyperglobulism. The superficial blood vessels, capillaries, and veins look full, so that the skin is always congested, in warm weather of a brick red color, in cold weather cyanosed. The engorgement of the face may be extreme, extending to the conjunctivæ, and in the cold the cyanosis of the face and hands may be as marked as any that is ever seen. There is often, too, a remarkable vasomotor instability, e.g., the hand becoming deeply engorged when held down, and rapidly anæmic when help up.

"The spleen is usually enlarged, but not to the great extent of leukæmia. It may vary in size from time to time. It is hard, firm, and painless.

"The total bulk of blood is enormously increased, and the ratio of corpuscles to plasma is high. The polycythemia ranges from seven to twelve or even thirteen millions of red corpuscles per c.mm. As a rule, they are normal in appearance and shape; mucleated red blood-corpuscles may be present, the hemoglobin ranging from 130 to 160 per cent., but the color index is relatively low. Moderate leucocytosis is the rule without any characteristic differential change; a few myelocytes may be present. The specific gravity is high.

"Of other symptoms the most common are headache, flushing,

and giddiness. Constipation is common, and albuminuria is usually present. The blood pressure is high; occasionally there may be hemorrhages into the skin and from the mucous membranes. Recurring ascites, probably in association with splenic tumor, was present in two of my cases."

In the American Journal of Medical Sciences of May 1921, E. P. Pendergrass reports two cases of his own and refers to the report of twelve other cases from the literature. In these cases the red cell count varied from 7,500,000 to 11,450,000, the hemoglobin from 110 to 120%.

November 13, 1922, Mrs. S. M. consulted me complaining of pain in her back, pain in her left side, hot flashes and sweats. She was a married woman, 49 years of age, and had two living children 29 and 24 years of age and had lost one child with diphtheria.

Three years previously she had had some operation through her vagina, when her uterus and a small dermoid cyst were removed by Dr. James E. King.

The record at the General Hospital showed that she entered there again December 29, 1920, and left there January 1, 1921. At that time she had apparently a plain case of chronic appendix with gastric symptoms. She declined operation and left the hospital.

She entered again September 18, 1922, complaining of a pain in her back and frequency and urgency of urination.

She gave the same history of operation and said that she had not menstruated since operation. At this time the interesting observations at the hospital were two blood counts:

9/20/22—Red 13,000,000, hemoglobin 100%, white 5,600.

9/22/22—Red 8,560,000, hemoglobin 129%, white 1,450.

In one count there were a few nucleated reds and an occasional myelocyte.

Her pulse rate averaged 80, temperature 98.6, blood pressure 124/102. Studies of her basal metabolism—9/22/22, 43.6% plus, 9/27/22, 20.3% plus.

At the hospital is this note.

Polycythemia vera.

1. Engorgement of superficial vessel, congestion of skin, face, conjunctivæ and hands.

2. Enlarged spleen, hard, painless.
3. Polycythemia 9,000,000—13,000,000.
4. Headache. Dizzy. Flushing. Constipation.
5. Hemoglobin high.
6. Vasomotor and functional disorders. Tingling, pricking, formication. Fluttering of heart.
7. Ocular disturbance—blurring of vision.
8. No elevation of temperature.
   No enlargement of heart.

At the time she consulted me she complained of dryness of mouth, the tendency to constipation, and of headache, flushing, hot flashes and dizziness.

Her pulse was 76, regular, blood pressure 196/90, temperature 98.6, and her weight 94¾ pounds, her greatest weight having been 150 pounds twenty years before.

Physical examination showed her tongue coated, marked pyorrhea with all of her teeth in very poor shape. There was no abnormality present in her nose, throat or lungs. The only abnormality about the heart was an accentuated aortic closure. Her abdomen showed no points of tenderness but her spleen was enlarged and hard and she said that she had pain there. There was no abnormal reflex. In addition to the suppuration in her gums there was also paronychia about the nails of her fingers. The urine showed a faint trace of albumin and an occasional hyaline cast. Her blood count was as follows: 11/13/22—Hemoglobin 100%, reds 8,496,000, whites 10,400, polys. 82%, small monos. 15, transitionals 2, eosinophiles 1.

An x-ray of her teeth showed the vital necessity of removing them all and she was so advised. She lived in Niagara Falls and went home with a few directions as to diet and medication.

She returned December 18th and at that time her blood count was as follows: Hemoglobin 109%, reds 9,536,000, bleeding time 1½ minutes, clotting time 3 minutes.

She had not yet had her teeth attended to so her bleeding time and the clotting time were taken and both found well under the normal limits. So she was sent home and urged to have her teeth attended to.

February 13, 1923, she came to Buffalo to have her teeth attended to. Her blood count showed: Hemoglobin 100%, reds 7,484,000, whites 13,400, polys 78%, monos 16, transitionals 4, eosinophiles 2. She had several teeth withdrawn but great difficulty was encountered in stopping the bleeding.

A week later she was put on benzol, five drops after each meal t.i.d. She had difficulty in getting this drug so did not really get started on treatment until ten days later.

April 4th her blood count was: Hemoglobin 96%, reds 8,064,000.

May 2nd she reported that on several occasions the benzol seemed to produce attacks of blurring of vision and dizziness. Her blood count showed. Hemoglobin 93%, reds 7,016,000. Her blood pressure was 170/100, pulse 78, temperature 98.3 and she weighed 95½ pounds. Her spleen was still palable, but not tender. The dose of benzol was reduced to four drops at a dose t.i.d.

May 22, 1923 she reported better in al respects. Headache abo it once a week and last two days but are not as severe and no heat flashes follow. Digestive tract was in all respects better and she had gained five pounds in three weeks. Her pulse 76, blood pressure 190/100, temperature 98.6. No cough but that of health. Physical examination: heart a little overactive; spleen still large, edge 1½ inch below costal margin but not hard or tardy. Blood: hemoglobin 90%, reds 7,464,000. Gradual increase in the dose of benzol was advised.

As regards the etiology, it is probably unknown, though several have ascribed it to tuberculosis of the spleen and a loss of function of destroying of the red blod cell. Others have ascribed it to a hyperplasia of the red bone marrow. The diagnosis may be made on the following facts: the great increase in the red cells and high hemoglobin, the peculiar color of the face—brick red when warm, intensely cyanotic when cold, the enlarged and hard spleen.

C. S. Williamson says, "The etiology is unknown, the prognosis is, in most cases, fatal, the treatment is thankless."

On the other hand several have been enthusiastic in praise of the treatment by stimulating doses of x-ray over the spleen, massive doses over the long bones and the use of benzol.

In the Journal of the American Medical Association, S. H.

Hurwitz and E. H. Falconer record the beneficial therapeutic results that have followed the combined use of benzene and roentgen rays in a woman, aged 38, presenting the classical signs and symptoms of polycythemia vera. "Although more than a year has elapsed since any form of treatment has been given, the patient's blood still shows a normal number of red blood corpuscles and a normal hemoglobin percentage. There has been an almost complete disappearance of the cyanosis of the facial prominences and extremities, and her general health continues excellent. In July 1916, the patient first came under the writer's care. A venesection was done and 500 c.c. of blood were removed. She was given iodids for a week. August 2nd roentgen-ray treatment was begun. October 9, 1916, she was admitted to the medical scervice of the University of California hospital, where she remained for two weeks. During her stay in the hospital she took 33 grams of benzene in one week. This was administered in capsules with equal parts of olive oil. A little less than five grams per day were given. After her discharge she took 8.5 grams of benzene from November 10 to 18, when the drug had to be discontinued on account of burning sensation in the stomach, with regurgitation and belching. From November 23 to 28, she ingested three additional grams of benzene. The drug now had to be stopped permanently on account of nausea, gastric discomfort and headaches. The only other form of treatment from September to November consisted of two roentgen exposures over the spleen, one of twenty-five milliampere minutes, and the second of thirty milliampere minutes. These were continued in December and January, five additional exposures of the spleen have been made during this interval. By the end of January 1917, the blood picture showed a complete return to normal, the patient's subjective symptoms had entirely disappeared, and the objective signs of the disease were decidedly less pronounced."

The authors are inclined to attribute this successful therapeutic result to the combined action of both agents. W. B. Baker, in the Journal of the Kansas Medical Society of July 1920, says:

"No treatment has proven uniformly satisfactory. Bleeding is practically always followed by temporary relief, benzol is perhaps

the most promising of all the drugs tried. The roentgen rays administered over the spleen have apparently helped in some cases."

During April 1923, my attention was called to another case in the Buffalo City Hospital.

Martin Rolka, 47, S. laborer. Two years ago noticed redness of face and occasional headache. June 1922 had a dizzy attack, slipped and had severe pain in his right side, since when he has had heat flashes and dizzy attacks.

He was excessively alcoholic until two years ago and smoked to excess, now no alcohol but still smokes fifteen cigarettes daily.

At present appetite, digestion and bowel action are normal. He has no cough and is not short of breath. He sleeps well but has occasional severe headache and dizzy attacks.

His pulse was 80, blood pressure 156/100, temperature 98.4.

Physical examination showed the tongue coated. The teeth showed some pyorrhea. The throat was normal and the nose was normal. The heart was a little large, A_2 plus, first sound slightly impure. Lungs normal. The abdomen showed the liver enlarged $2\frac{1}{4}$ inches below the costal margin, $6\frac{3}{4}$ inches over all, the spleen six inches, palpable and hard, one inch below costal margin, slight tenderness in right iliac region, no masses, no hernia, and no edema of legs. Knee reflexes sluggish, no ankle clonus and no cremasteric.

Urine 1500 c.c., specific gravity 1018, acid. Albumin two plus, sugar 0, bile 0, urobilin 0. Many hyaline and granular casts.

Blood: Hemoglobin 160% Sahli, reds 11,480,000, whites 10,000, polys. 70%, small monos. 10, large monos. 7, transitionals 10, eosinophiles 1, basophiles 2. Wassermann, negative. Basal metabolism, plus 17.

I have seen this case but once. As it is to be reported by the attending physician, Dr. A. Harrison, I will only further report that he has been used as a donor in several cases needing transfusion. He has been made temporarily better by each bleeding, yet it has been noticed that the blood of the patients receiving blood from this man has showed a better and more prolonged improvement than usual in cases following transfusion.

PERSONAL EXPERIENCE WITH THE USE OF ILETIN (INSULIN) IN THE TREATMENT OF DIABETES MELLITUS.

By JOHN A. LICHTY, M.Ph., M. D.,
PITTSBURGH, PA.

Introduction.

In May, 1921, a group of men organized and directed by Prof. J. J. R. Macleod, Department of Physiology of the University of Toronto, undertook to separate from the pancreas the internal secretion of the islands of Langerhans. This undertaking was instigated by the conclusions arrived at by F. G. Banting, then an assistant in physiology at the Western University, London, Ontario. Banting from certain suggestions which he picked up in the medical literature pertaining to diabetes and its particular relation to the pancreas, conceived the idea of separating the external portion of the gland from the internal portion, and thus obtaining the internal secretion and making it available for the treatment of diabetes mellitus.

The use of this internal secretion of the islands of Langerhans (*Insulin*) in the treatment of diabetes mellitus has been under observation by the clinician now for about one year, though its general use, under certain restrictions, has been established only for about the past four months.

Scope of Discussion.

It might seem premature, therefore, to report any definite results from this plan of treatment. But my objective is not to attempt a statistical presentation; it is rather to speak of my personal experience with a comparatively small group of patients in whose cases the product insulin was administered. No doubt the stamp of approval, or disapproval for that matter, and the extent to which this treatment will be applicable will come from, and will be determined by the many similar reports of cases which will be offered

from time to time in the next few years. *Animal* experimentation must be confined to the laboratory from which the reports concerning this substance have come and are still coming, but *clinical* experimentation and observation must be reported from the places where these patients live or may be induced to congregate. Some of these reports may be of great and others no doubt of very little value, depending upon the comprehension of the problem and the reliability of the observations made by the individual reporting them.

Great credit must be given to the Toronto group of investigators for keeping this work from newspaper publicity, and for keeping the administration of the remedy for a time in the hands of the few whose qualifications were known personally and upon whom they could rely for that gradual introduction to a larger field which any new remedy deserves. This protecting guidance in itself furnishes an interesting chapter in the history of this discovery.

Note: I wish to express my appreciation personally to the Toronto group for making it possible for me to obtain insulin for my earlier cases, and also to thank Eli Lilly & Company for their willingness to supply me with iletin, gratuitously for a time, for subsequent cases, as well as for their promptness and helpful suggestions.

The impatience which has been manifested by the laity, and by the profession as well, from time to time, with reference to the difficulty of obtaining insulin cannot be wondered at when one recalls the large number of people who are suffering from diabetes and who are without hope of recovery, and also when one recalls the difficulties and disappointments with which the physician has had to contend heretofore when he undertook the treatment of these patients.

Former Methods of Treatment.

In my own experience I have seen in the past thirty years approximately five hundred patients with diabetes mellitus. A few of these may have been only cases of simple glycosuria. A review of the lines of treatment adopted from time to time in these patients brings out clearly the fact that thirty years ago we were about at the end of the so-called "medical treatment" period. It was a failure, as no specific remedy could be found in the pharmacopœia. I have in my possession some very interesting diagramatic charts which were sup-

posed to show the effect of the "codeine treatment" in a severe case of diabetes in a patint in the medical wards of the Philadelphia Hospital in the winter of 1894-95. It was remarkable the amount of codeine this patient could take with safety when the dose was increased gradually. The urine diminished in volume, and the sugar in amount, but the caloric value of the diet could not be increased, in fact had to be diminished, and the patient died in coma.

The period following that of specific medication might be called the period of "high caloric feeding." Carbohydrates were practically eliminated from the diet forthwith, when it was possible, but high fat and high proteid feeding was substituted. Von Norden and others advocated specific carbohydrates, such as oatmeal, potato, rice, etc. This treatment was based upon the physiology then known, but the failures and the cases of diabetic coma were, if anything, more frequent.

Next came the period which promised more for these hopeless individuals than any previous plan of treatment. This began about 1912 and was the result of the epoch-making work of Dr. Frederick M. Allen. He produced the disease, diabetes mellitus, in dogs and then placed those same dogs upon a diet characterized by a low caloric value and controlled the glycosuria with remarkable ease.

From this time on the patient with diabetes mellitus became more hopeful. The number of deaths from coma diminished, and the complications were not so frequent and not so dangerous. Too much credit cannot be given to Allen for his careful work. The practical clinical application of this is typified in the common-sense way in which Dr. Elliott P. Joslin applied it in his own work, which has since become the guide of many physicians and dietitians and patients.

We now enter into the fourth period, that of a specific remedy representing the internal secretion of the islands of Langerhans. This seems most rational, and has been looked for for a long time, or ever since it was shown that the active principle could be separated from any gland of internal secretion. No doubt it is largely due to the recent development of physiological chemistry that this discovery was made possible at this time. Acknowledgment has already been made to J. B. Collip of the Department of Pathological Chemistry of

the University of Toronto for his share of the work in relation to the problem of isolating the active principle "iletin" and of freeing it from the objectionable proteids.

In conformity with this brief review my 490 cases naturally divide themselves into four groups as follows:—

(1) Those few in which it was attempted to use a specific drug. The results were necessarily disastrous.

(2) Those (186) in which it was attempted to eliminate the carbohydrates, excepting certain specified forms, from the diet and increase proportionately the proteids and fats. The results were somewhat encouraging.

(3) Those (242) in which the Allen and Joslin plan of resting the pancreas, by a certain reduction of the diet and then later gradually increasing the diet, was adopted. This gave such satisfactory results that one felt justified in holding out new hopes to the diabetic. Some of these cases were of patients who have become sugar free, and now living a normal life, and are apparently well. These favorable results were usually in patients at or beyond middle life.

(4) And finally (27) those in which *insulin* is now employed, the results of which I shall briefly narrate. These twenty-seven patients were selected from forty-four patients with various forms of glycosuria which presented themselves for treatment since about November 1, 1922.

Report of Cases.

My experience with insulin began in a rather indirect way early in August, 1922. I had at that time under my care a patient whom I saw first in April, 1920, and whose history is briefly as follows:—

Case 1. Miss J. T., No. 21,764, aged five years, had been in good health, excepting that she was subject to occasional attacks of pain in the abdomen with nausea and vomiting. No cause could be given for these attacks. Two years ago she had three or four attacks of tonsillitis in rather rapid succession, after which the attacks of abdominal pain no longer recurred. Three weeks before consulting me she became unusually thirsty and had polyuria. Six days ago sugar was found in the urine. Her weight was 42 pounds. There were no abnormal physical findings, except that she had diseased tonsils. On a general diet her blood sugar was 0.14 and the amount

of sugar in the urine was 12% in 1050 c.c., or 126 grams in twenty-four hours.

She was sent to the Mercy Hospital and placed upon the Allen-Joslin treatment with certain modifications. A radiologist treated the tonsils. The child coöperated very well and in a reasonable time she was sugar free on about 700 calories. After leaving the hospital sugar occasionally returned in the urine, but with a slight modification of the diet it could be eliminated.

In October, 1921, she was accidentally exposed to diphtheria, and two weeks later had a sore throat which fortunately, however, was not diphtheritic. After this she had an attack of rheumatic fever from which she made a recovery without any heart involvement. But from that time on it was difficult to keep her urine sugar free. Her diet was reduced from time to time, and in August, 1922, she was very ill. She weighed less than 38 pounds; her blood sugar was 0.28; daily urine 3720 c.c. with 1.2% sugar (44.64 grams) on practically no food intake. She had a general ædema and was listless. It was thought she could not live. We then called the attention of the Toronto Clinic to her case through the kind offices of Dr. R. H. Henderson of Kitchener, Canada, a close friend of the family, and on August 19, 1922, she was taken to Toronto. She was carried to the clinic as she was unable to walk. I am unable to give the details of the treatment there, but insulin was given and under gas anæsthesia, at an opportune time, an enucleation of the tonsils was done. She returned to Pittsburgh, October 19th, after just two months of treatment, weighing six pounds more than when she left her home and taking thirty grams of carbohydrates, thirty grams of proteids, and ninety grams of fats. Her cheeks were rosy and her body was plump. She was taking about fifteen units of insulin hypodermatically before each meal. There was occasionally sugar in the urine.

October 31st I was advised from Toronto that Eli Lilly & Company would supply me with insulin for this patient under the trade name of "Iletin," which I have since used. With the use of the first shipment there was a slight protein reaction, but since then the preparation has been satisfactory throughout. The patient is going to school half days now and seems and acts like a well child. At

present she weighs fifty-five pounds and is growing in stature. Her diet contains approximately thirty-five grams of carbohydrates, thirty-two grams of proteids, and 140 grams of fats. To metabolize this she has administered hypodermatically fourteen units of Iletin before breakfast, eight before lunch, and fourteen before dinner. This patient's life was undoubtedly saved by the use of insulin. I am attempting to diminish the Iletin now, and her improvement is continuing.

In another case which began the treatment in December, 1922, I succeeded in gradually withdrawing the Iletin until the patient got along without any. This remarkable case deserves to be reported because it opens up the large question of the permanency of the effect, or in other words the possibility of a cure of the disease.

Case 2. Mr. J. W. K., No. 23,284, aged 16, a school boy, consulted me December 12, 1922. His symptoms began about October 27th, 1922, without any apparent cause. His father's uncle and a cousin on the father's side had diabetes. The physical signs were negative. His weight was 96. The urine contained 6% sugar and the blood sugar was 0.19. By gradually withdrawing the fats and then the proteids, and later reducing the carbohydrates the urine became sugar free in seven days and the blood sugar came to 0.08. The diet contained about 363 calories then. It was increased rather rapidly and Iletin was given before each meal. It will be seen by the chart that this patient's urine remained sugar free from the first. Later, because the blood sugar remained low even though the food was increased, the Iletin was diminished. January 17, 1923, on account of our supply of Iletin being for the time exhausted, it was withdrawn entirely. No sugar appeared in the urine and the blood sugar was 0.1. This patient has been seen at intervals since. His blood sugar has never been higher than 0.14, and the urine has remained sugar free. His diet at present is 120 grams of carbohydrates, 100 grams of proteids, and 200 grams of fats. He now weighs 116 pounds, a gain of twenty pounds, and has grown three-fourths of an inch. He has returned to school. This patient is naturally being watched with great interest. On April 24 the urine was 1020, normal, 1250 c.c.; the blood sugar 0.10 on a diet of 120 grams of carbohydrates, 100 grams of proteids, and 200 grams of fats. I

understand similar cases have occurred in some of the other clinics, but as yet I have seen no published report.

This is the first patient I have treated with Insulin which had not had a previous trial with dietetic treatment only. The contrast of the two treatments cannot be so definitely shown, therefore. The next case I shall report shows the contrast very definitely. It is in an adult patient who has been under my care since June, 1920. Since that time she has been in the Columbia Hospital two different periods. During the first period she did very well. For almost two years thereafter she increased her diet and improved, but on account of a sudden bereavement her tolerance broke down and she returned to the hospital in May, 1922. The results at this time were not so good. She was later sent to the diabetic clinic of Dr. S. T. Nicholson of Clifton Springs Sanitarium when she did better for a time, but later her tolerance diminished so that she apparently could not metabolize more than about 80 grams of carbohydrates. October 9th, 1922, she was taken to the diabetic clinic of Dr. John R. Williams at the Hiland Hospital, Rochester, N. Y., where Insulin treatment was undertaken. It is only fair to Dr. Nicholson to state that he had just received his first consignment of "Iletin" from Eli Lilly & Company as this patient was leaving for Dr. Williams' clinic. The history is as follows:—

Case 3. Mrs. J. W. F., No. 20,463, æt. 42, consulted me first in June, 1920. Sugar had been found in the urine four weeks previously. The patient has not been well since an atttack of influenza during the winter of 1918-19. Five weeks ago she first noticed excessive thirst and polyuria.

The physical examination showed a loss of weight from 128 to 111 pounds. There is an adenomatous thyroid, but no definite thyroid symptoms could be elicited. There is also a general visceroptosis.

The urine contained a heavy trace of suagr and the blood sugar was 0.23. She was sent to the Columbia Hospital and her subsequent history was as described above. Several factors entered into this case which needed consideration. The patient began to develop occasional manifestations of thyrotoxicosis and she was also passing

through the menopause. Occasionally there was great mental depression.

Dr. Williams reported to me that the patient's weight was 69 at first while at the Hiland Hospital. With the use of Iletin and on a proper diet the weight came up to 99. When she left the hospital she was taking 52 grams of carbohydrates, 27 of proteids, and 180 of fats. The urine occasionally showed sugar. At present his patient weighs 114 pounds. She now takes 54 grams of carbohydrates, 84 of proteids, and 181 grams of fats, and needs 35 units of Iletin to keep the urine sugar free.

Case 4. Mrs. S. S., No. 9,882, æt. 52, consulted me in 1910 on account of obesity and pain in the knees. The patient was much above the normal weight. The knees presented no physical signs suggestive of pathology. The urine was normal. It was concluded that the pain in the knees was due to excessive body weight and to faulty posture. A reduction diet was established and with the decrease of weight the symptoms disappeared.

The patient then passed from my observation until July, 1918, when she appeared with an increase of weight and the return of her former symptoms. One per cent sugar was then found in the urine. With a limitation of the carbohydrates of her diet the sugar could be easily eliminated from the urine, and for the following four years the patient was in comparatively good health. There was considerable arterial change for a sixty year old individual. The blood pressure was 150-100.

In the fall of 1922 the patient entered the Mercy Hospital because of the gradually diminishing carbohydrate tolerance. The urine was 1020, acid, 2750 c.c., and 2% sugar. The blood sugar was 0.28. Under the usual Allen-Joslin regime the patient's urine became sugar free and the blood sugar came down to 0.08. The intake was only about 1200 to 1400 calories. About this time Iletin was available and in December this treatment was begun. In this case it was possible to advance the diet to 1800-2000 calories by administering fifteen units once a day. The intake was approximately 130 grams of carbohydrates, 70 grams of proteids, and 140 grams of fats. It was attempted several times to increase the fats and also to increase the Iletin, but each time the blood sugar went

up and sugar appeared in the urine. The patient feels better, looks better, and has the satisfaction of eating a more varied diet. Her carbohydrates are no doubt higher than they should be, but she prefers her diet as it is even though it requires more Iletin.

Case 5. Mrs. I. B., No. 23,857, æt. 62, was referred to the Medical Department of the Mercy Hospital from the Orthopedic Department by Dr. James O. Wallace. She entered the hospital on account of a fracture at the neck of the left femur due to a fall. In the course of the examination 4% sugar was found in the urine. A cast was applied and the patient was transferred. She had a right-sided facial paralysis, due evidently to a swelling in the right parotid which she said had been noticed for five years. There was also a swelling of the knees like a vilious arthritis. The patient was of the obese type, and an attempt was made to reduce the sugar in the urine by leaving off the fats first, later the proteids, and finally reducing the carbohydrates. The blood sugar was 0.25 on admission. The urine became sugar free and the blood sugar fell to 0.10. At this point a profuse diarrhœa began which suddenly dehydrated the patient. The signs of coma appeared and developed rapidly. Water was given by mouth and subcutaneously. The blood sugar increased to 0.25. In two days eighty units of Iletin were administered. The blood sugar came down after each administration, but immediately went up again. The coma became deeper and death followed at the end of four days. This case presented insuperable difficulties on account of the pain from the fracture, the discomfort from the cast, and possibly not the least from our own inexperience with such complications.

Another case of diabetic coma in which Iletin was given died under my care at the Mercy Hospital. This patient had an old, unrecognized, suppurating mastoid which gave symptoms of cerebral pressure. Her diabetes has been of long standing. She was emaciated and showed signs of beginning coma when she entered the hospital. It was rather difficult to determine how much of her condition was due to mastoid disease and how much to diabetes mellitus.

In both of these cases the Iletin showed its value in controlling the hyperglycæmia and the glycosuria, but one was soon impressed with the fact that the treatment of diabetic coma meant more than

controlling the hyperglycæmia and glycosuria. In both cases it was difficult and finally impossible to keep up the necessary supply of fluid for the body, though all the avenues for getting fluid in were utilized. It goes without saying that finally nutrition failed also, and the Iletin seemed to bring about only a disappearance in the blood and urine of the evidence of the marked katabolism which characterizes diabetic coma. Twenty-seven cases have now been treated with the aid of Iletin. They do not constitute the whole number of patients presenting diabetes mellitus and glycosuria seen since the Iletin treatment was begun. These five patients were reported as representing certain types of the disease. These types appear as follows:—

TYPES OF CASES.

Type I. The young patient, the child, who has definite fixed diabetes, and who needs continuous Iletin administration to maintain health and efficiency.

Type II. The young patient with symptoms of only short duration but definitely those of diabetes mellitus in whom the Iletin seems to set aside the symptoms entirely, and there is apaprently continuous health and efficiency without further administration of Iletin. I have thus far had only one case, but I have heard of others and I feel that probably more will be found when heads of families will see that the urine of their children is examined systematically and an intensive treatment, including the use of Iletin is immediately undertaken.

Type III. The definite adult diabetic who cannot handle sufficient calories to maintain life at a certain degree of efficiency without Iletin.

Type IV. The old obese patient, frequently also arteriosclerotic, who often chafes under the restrictions of a diet and wishes more leeway. This is the type which is likely to develop gangrene. It is not unlikely that these patients will be saved from such a complication. In two of my patients of this type who already had well developed gangrene of the toes the use of Iletin undoubtedly favored healthy granulation and extensive amputation was not necessary.

Type V. The case presenting the symptom of diabetic coma. This is not a distinct type in itself as coma may occur in any

diabetic, but it is more likely to occur in types one and three. From the reports of other observers I infer that the Iletin treatment is more likely to be successful in types one and three than in type four. I can certainly bear testimony to the impression that in type four, coma, while it may not occur so readily, when it does occur it is more difficult to overcome, and Iletin does not seem to fill the purpose as it does in other cases.

Limitation of Treatment.

I have already suggested that not all patients having diabetes require Iletin treatment. This statement should be preceded by one declaring that not all patients presenting a glycosuria require Iletin treatment. The adult, with the transient glycosuria and the nephritic case with glycosuria, need no such treatment. In fact great harm and danger may arise from an attempt to use Iletin in these cases. In the true diabetics the treatment is necessary only in those who continue to have sugar in the urine, or to have a blood sugar above 0.15-0.17 on a diet furnishing less than approximately thirty calories per kilo. To use it in other cases at the present development is somewhat of a luxury, especially in cases of adults. In children the case may be different, as has already been suggested.

The reason for speaking of it as being a luxury in the above defined cases is because the substance is still rather expensive—four and one-fourth to seven cents a unit. Sometimes as much as fifty to seventy units a day are necessary. Besides, unless the patient or a member of the family can be safely entrusted to administer the hypodermic, the expense of a nurse or a physician who can come to the patient one to three or four times a day must be added. It must also be remembered that the diabetic who becomes an Iletin patient can scarcely do so before first becoming a hospital patient, because a hospital organization, including skilled laboratory workers and experienced dietitians with a director of that department, seems necessary, with our present knowledge and experience. The "carbohydrate tolerance" and the required and safe dose of Iletin necessary for each individual patient must be established in a hospital. Already hospitals and private institutions are finding that the diabetic department does not cover expense unless a super charge

is made in that department. It is possible with additional knowledge and experience the method of treatment can be simplified so as to make it less exacting and less expensive. It is encouraging to know that men of large experience in this work, such as Joslin of Boston, and Campbell of Toronto, are keen to this situation and already are suggesting more simple methods. Unless this point is recognized there must be established heavy endowments of centers where this work can be carried on at a nominal expense so that the treatment may be available for the middle class and for the poor, and also for those without any resources whatsoever.

Plan of Treatment.

The plan of treatment with Iletin which has been gradually developed by my colleagues and myself at the Mercy and the Columbia Hospitals is about as follows: The patient is required to come to the hospital for from three to four weeks. If the case is a severe one the time may be longer. We are hoping and striving to make it shorter. Daily collections of the urine are taken, and the blood sugar is estimated once or twice a week, or as often as the exigencies of the case demand. The diet to which the patient had been accustomed is continued for one or two days, and roughly estimated as to the number of grams of carbohydrates, proteids, and fats. The weight is taken. The patient is not allowed to remain in bed the entire twenty-four hours of the day, unless he is too weak to be up. He is encouraged to go out doors and to take a prescribed walk. We have long ago learned the necessity for such care. When the laboratory data is at hand such changes are made in the diet as we formerly used in carrying out the "Allen-Joslin Method," as we have been accustomed in the past ten years to denominate it.

On the day the patient's urine becomes sugar free the blood sugar is estimated. If this is low, that is, at or below 0.10 or 0.12, the diet is carefully increased until a trace of sugar appears in the urine. Iletin is then given hypodermatically about one-half hour before each meal, beginning with from three to four units at a dose. If, when the trace of sugar appears in the urine, the caloric intake is as much as or more than 30 per kilo, it is usually decided that Iletin is not necessary. This decision may depend on certain

economic and other factors. If the patient's means are limited, and especially if it is thought that it will be rather difficult to carry on the treatment at his home later on account of distance from he hospial or of difficulty to establish satisfactory relations with the family physician, Iletin is not begun unless later experience proves that the case is more severe than was first thought. If, on the other hand, the patient does not become sugar free on the Allen-Joslin treatment, Iletin is begun with a very small dose, perhaps one to two units given hypodermatically before each meal, and gradually increased. At the same time the diet remains fixed for the time at the value already determined. As soon as the urine becomes sugar free the blood sugar is estimated, and *accordingly the diet and the Iletin are increased or diminished together or separately*. It must always be kept in mind that a reduction of diet or an increase of Iletin may bring on the grave symptoms of hypoglycæmia. There is a certain feeling of restlessness, internal tremor, and oppression beneath the sternum. There is shortness of breath and anxiety. If the hypoglycæmia is decided, such as 0.05, there may be convulsions. These symptoms will subside promptly by having the patient take the juice of an orange or plain sugar. When the blood sugar is high, that is 0.17 or more, there is of course very little danger of producing hypoglycæmia with a single dose of Iletin.

The plan which we have adopted may not be the most expeditious, but we feel it is safe, and in the long run is satisfactory.

Our plan is to get the patient as nearly as possible upon a diet which represents about thirty to thirty-five calories per kilo of body weight, depending upon whether it is a growing child or an adult, with as little Iletin as possible, and given in the smallest number of doses. In some cases one dose a day is sufficient; in others four does a day are necessary, the fourth being given after midnight. At the same time we aim to keep the urine sugar free and the blood sugar below 0.17. The gratifying experience in the use of Iletin is that even if these ideal conditions cannot be established, even though the blood sugar remains at the high level of 0.17 or higher, and sugar continues to appear in the urine, if the carbohydrates are increased in the diet, and if sufficient Iletin can be administered to metabolize it, the

patient will improve apparently just as satisfactorily as if he was sugar free. This I have noticed in several of my patients.

The most difficult part of the whole plan of treatment is necessarily that part which follows after the patient leaves the hospital or one's direct supervision. Before the use of Iletin, it had become quite possible to instruct patients about food values and how to test their urine for sugar. In this way a "home treatment" could be fairly successfully carried on. To this regime must now be added the antiseptic handling of a hypodermic syringe. Some one, either the patient, a member of the family, a nurse, or a physician must be detailed to inject the serum, and certain instructions must be given with reference to the symptoms of an over-dose of Iletin and the treatment to be established when an over-dose is given. I have not adopted a definite plan as yet to meet these added requirements. But with nearly every patient leaving the hospital or my immediate care, thus far satisfactory arrangements could be made. Either the family doctor came to the hospital and got the necessary information, or a nurse or a member of the family undertook the work. In some cases the patient prefers to administer the hypodermic himself. It is no small task to see that the serum is at hand for each patient at the right time and in sufficient amount. In my own work thus far this is done through my office. Weekly shipments are received from Eli Lilly & Company and the necessary amount is distributed to each patient from time to time. I here wish to acknowledge my indebtedness to my office nurse, Miss Traphagen, who has taken this part of the work in hand and is carrying it on so successfully. By correspondence and by having the patients report in person from time to time rather satisfactory contact has thus been kept up.

Results.

What are the present results arising from the use of Iletin and what will be the ultimate results? In my own experience it is plainly evident that a patient's carbohydrates tolerance may be elevated by means of Iletin in a way that I have never experienced before. The patient gains weight instead of losing, and he is physically and mentally more efficient. Though it has happened that my experience in many cases of diabetic coma has not been satisfactory, I believe it is

of great value in this condition. I am informed by others that the young patient or the adult without complications is more successfully handled with Iletin in diabetic than the old complicated cases, such as my two unsuccessful cases were. Of one thing I am sure and that is that the horror of diabetic gangrene is now a thing of the past.

As to whether an ultimate cure can be expected in certain cases, and as to how long the treatment must be or can be safely carried on, our present short experience does not warrant a definite answer.

In conclusion I wish to express my indebtedness to the office and hospital force, Wm. A. Bradshaw, M.D., John O. Woods, M.D., Miss Emma Traphagen, R.N., Mrs. Miel, Laboratory Technician, and Miss Crawford, B.S., stenographer, all of whom added their labor and enthusiasm to this time-consuming work.

DISCUSSION.

Dr. Robert Wilson, Jr.: I wish to refer to two or three of my cases.

One had been under my care for only about two weeks. When I first saw her the blood sugar was 519 mgrs. which rose in a day or two to 533 mgrs., and the urine sugar was 132.65 grms. in twenty-four hours with a large amount of acetone and diacetic acid. She was put on fifteen units of Iletin before each meal and the diet restricted as usual. The disappearance of acetone and diacetic acid was almost immediate and was quickly followed by a disappearance of the urine sugar and marked reduction of the blood sugar.

Another patient was an old diabetic with a severe case of broncho-pneumonia. At the time treatment was begun the pulmonary condition had improved, but there were still very pronounced signs of infiltration and the general condition was bad. The blood sugar was 285 mgr. and the urine sugar 90 grm. in twenty-four hours. He had already been carefully dieted. Iletin was given in fifteen unit doses. Response was not as prompt as in the first case, but at the present time the urine has been sugar free for about two weeks and the blood sugar has fallen to 162 mgr. The lungs are entirely clear and he has gained considerably in weight. In view of the seriousness of acute infections in diabetes the results in this case are very significant.

A third case illustrates the value of Insulin in acidosis. The patient was advanced in years and was a diabetic of long standing. She developed very pronounced symptoms of acidosis with marked somnolence which failed to respond to the usual treatment. Quantitative estimations of blood and urine sugar could not be made. Her response to Insulin in the same dosage as used in the other cases was prompt, the symptoms disappearing completely in a day or two. She proved to be a very refractory patient and would not coöperate so that it was impossible to accomplish more than the relief of the dangerous symptoms.

In Insulin we have, undoubtedly, an agent of very great value, but I do not think it has been in use long enough for us to determine the extent of its usefulness and its limitations.

Dr. Pritchard: At our clinic we have treated some fifty patients of diabetes with Iletin. In each case the extract is administered for three months or longer. We are greatly indebted to Dr. Banting for his help in guiding our earlier endeavors in this therapeutic field. On account of the scarcity of the product it was necessary for us to use a home manufactured variety. This required a great deal of time and care. Regardless of the lack of refinement in the earlier preparation, the results of its use were most encouraging.

All the cases were institutionally treated and Iletin was used as an adjunct to former methods. From our experience with this treatment we may mention the following points as of special interest:

(1) Children respond readily and favorably to this specific treatment.

(2) All cases of diabetes do not require Iletin administration.

(3) Cases of coma or moderate acidosis often show the most imrpovement.

(4) Gangrenous tissue responds readily to this treatment. The process is often retarded and cleared by the use of Iletin.

(5) Neuritis so frequently associated with diabetes is often checked after the administration of the extract. This may be due to the reduction in the blood sugar.

(6) Caution must be used in this treatment. Repeated blood sugar estimation and urine analysis should be recorded.

Dr. Lichty: With reference to other complications such as gangrene. I have a patient who has gangrene of both feet. He should be operated upon, but has refused. He has saved all of his toes. With the use of Iletin I can keep him going indefinitely.

The question of expense is of vital importance. I think a traveling dietitian, to get the specimens of urines, etc., is really a helpful suggestion.

We do not know whether a patient can take this Iletin for five or six years.

ABSCESS OF LUNG.

By JAMES ALEXANDER MILLER and ADRIAN V. S. LAMBERT,

NEW YORK CITY.

Introduction.

This report is based upon our experience with sixty cases, mostly seen in the last two years. The comparatively large number of cases observed during so brief a period appear to indicate that abscess of the lung is a more common condition than is generally appreciated.

A study of the literature indicates the existence of considerable confusion in the reports on various suppurative lung conditions. There would appear to be need for greater accuracy in definition between acute pulmonary abscess, chronic pulmonary abscess, so called gangrenous abscess and pulmonary gangrene, bronchiectatic abscess and bronchiectases.

The differetial diagnosis is not always easy, as our best means of arriving at this is the x-ray and the pictures presented by acute abscesses and acute exacerbations engrafted on chronic underlying lesions may be quite similar. The histories in many cases may lead us to suspect the existence of a chronic etiological factor but often it may be impossible to obtain the facts, and yet the importance of making this distinction is obvious as proper treatment depends largely upon its accuracy.

The series here reported has to deal mainly with acute pulmonary abscess, forty-six of the sixty cases being of that nature.

Etiology.

It is generally considered that pulmonary abscess follows a pneumonia in which necrosis and liquifaction supervene. It is usually considered a primary broncho and lobar pneumonia or one secondary to other conditions such as operations on the upper air

passages, aspiration of foreign material, traumatism to the chest, pyemic metastases from suppurating foci elsewhere in the body.

Our experience leads us to surmise that the condition of abscess of the lung may occur without any evidence of preceding pneumonia. Such a condition may be supposed to be an abscess primarily from the start, the interstitial tissue of the alveoli being infected and the inflammatory products breaking down promptly without any definite stage of an exudative pneumonia into the air vesicles.

In our series such primary abscesses would appear from the history to have been a probability in twenty-two of the sixty cases.

The history of such cases is a rather short and not very acute prodromal period which develops in a short time, usually within a week, into the definite symptoms of an abscess discharging into a bronchus, evidenced by the expectoration of considerable quantities of pus.

In favor of such an hypothesis of primary abscess formation is the fact that secondary abscesses in the same or opposite lung not infrequently occur. That these secondary abscesses have the same character as the original lesion, without signs or symptoms of a preceding pneumonia, and following the inhalation of foreign bodies such abscesses are well recognized, independent of any intermediate pneumonic stage.

This primary abscess formation may occur either because of local favoring factors in the lung itself or because of the nature of the infecting agent.

Among these local factors in the lung itself the irritant action of ether vapor should be considered as one. The incidence of post operative pneumonia following its inhalation and its deleterious effects on those suffering from preëxisting pulmonary lesions but confirm this view. As to the nature of the infecting agent, our observations upon the bacteriology may have a bearing upon this question.

The usually accepted idea of the bacteriology of lung abscess is that of a very mixed group of pyogenic organisms upon which may be engrafted the putrefactive organisms in the case in which gangrene appears.

Our studies of the bacteriology are limited to only ten of our

cases in which the examinations were made directly from the abscesses at the time of operation. They do not include any studies of the sputum, for these are unreliable on account of the ready liability to contamination with bacteria from some other portion of the respiratory tract.

The striking results of these bacterial examinations of the abscess pus in these ten cases were the uniform presence of anærobic bacteria which were present in predominating numbers in all and were the only type of organism found in eight of the ten cases.

In the other two cases streptococci were found, one a hemolytic and the other a non-hemolytic variety, in addition to the anærobic bacteria.

The anærobic flora found were quite variable, being a streptothrix in six cases, a Gram-positive coccus in six, a Gram-positive bacillus in four, a Gram-negative bacillus in seven, a fusiform bacillus in two and a spirillum, closely resembling that in Vincent's angina, in one case.

It was interesting to note that in one of our cases successfully operated upon for abscess in the right lower lobe two anærobic organisms, a streptothrix and a Gram-negative bacillus were the only bacteria found, and that sixteen months later after the patient had become apparently well, another pulmonary abscess developed in the right upper lobe and from which the identical two organisms were again the only ones isolated from this abscess at the second operation.

We have attempted some preliminary experiments with monkeys in the endeavor to produce lung lesions by intratrachial injection of these anærobic organisms and in one monkey this was done in conjunction with tonsillectomy. The animals reacted with some fever for a few days, but no lung lesions were produced.

We shall continue this study as it appears to us to have an important bearing on the subject.

The fact that some of these anærobes correspond to the type found in the mouth and throat in such conditions as pyorrhea and Vincent's angina raises the further interesting query whether the well recognized relationship between pulmonary abscess and operations upon the upper air passages may not, in part at least, be due to the

pathogenic possibilities of these anærobic bacteria in producing abscess when they reach the lung under the varying conditions of the inspiration of foreign infected material at the time of such operation.

Methods of Treatment.

The cases of this series with four exceptions have been studied jointly from the medical and surgical points of view, the hospital cases which compose the larger part being the subject of a medico-surgical conference held at least once a week during the entire period of their observation. This we believe is the ideal method of investigation.

Originally we approached the problem of their treatment as primarily a surgical one and in the earlier part of our studies we were inclined to advocate early operation as soon as the diagnosis was accurately established. These operations were performed independent of the age of the patient, his condition or the type of lesion present. The results were most disheartening and consequently about one year ago we radically changed our procedure by treating them primarily as medical cases, advocating operation only after medical treatment had failed or after the local conditions for successful operation had become more favorable.

The medical treatment consisted of rest and postural drainage, the latter carried out regularly four or five times a day for periods varying from five to twenty minutes each.

Postural drainage is carried out by inverting the patient over the side of the bed with the head down at the floor. This position is maintained for three to five minutes at first and repeated four or five times a day. As the patients become accustomed to it the maintenance of this posture is increased in duration to as long as may be necessary to thoroughly empty the abscess cavity. This is usually not more than fifteen or twenty minutes at each session and is frequently less. The patient soon comes to recognize the period desirable in his individual case and also can appreciate the need for the repetition of the procedure by the returning tendency to cough and expectorate. When successful this method affords several hours of almost complete freedom from cough and expectoration, is accompanied by marked diminution in the constitutional symptoms and

after a few days by a gradual diminution of the total quantity of the expectoration as well as of its fetid purulent character.

We believe that by this method it is possible to increase very materially the number of cases which may be cured by spontaneous drainage through a bronchus. It also will aid in diminishing the extent of the area of inflammatory reaction in the lung surrounding the abscess and also improve the general condition of the patient and render the case more suitable for operation should this become neecssary.

The surgical treatment consisted in the main of incision and drainage in one stage if the pleural cavity was walled off by adhesions, otherwise two stages. Local anesthesia is certainly safer than general anesthesia even with nitrous oxide gas and oxygen. Soft rubber dam we believe the best form of drain, and have abandoned the use of gauze, employing it only to control hemorrhage. We found this operation extremely dangerous and usually unsuccessful in the acuter phases of the disease, that is, during that period when there was present about the softened central suppurating focus a more or less extensive zone of œdematous soggy infected lung in which the smaller bronchi are filled with pus and the alveoli are distended with sero purulent exudate and while the walls of the alveoli are swollen and œdematous. None of these cases were seen prior to their rupture and communication with a bronchus.

Our experience has led us to believe that incision of the abscess at this time fails to effectively drain the entire infected area and that with a free opening established the effectiveness of coughing in emptying these areas is greatly reduced. In many of our cases opened during this stage there was a pronounced improvement for a few days, the expectoration was diminished, the temperature became normal and the patient apparently was making a satisfactory recovery. But this period was followed by a wide extension of the pneumonic process, sometimes ushered in by a chill and a fatal termination in from three to five days or a week. At autopsy there was found a widespread massive involvement of the entire lung or both lungs in which the bronchi were filled with pus and the alveoli either showed the changes of an acute pneumonia or with alveoli swollen and œdematous and infiltrated with leucocytes. In some cases which

lived long enough there were multiple small abscesses widely disseminated throughout the lungs.

After postural drainage, however, even if it does not effect a cure in itself, there is usually a definite improvement in the constitutional symptoms and a clearing of this secondary zone of infiltration about the abscess, which renders an operation much more liable to succeed.

There is a period in lung suppuration when the alveoli in the immediate vicinity of the abscess are collapsed but with the respiratory epithelium still intact and when they are therefore capable of reæration, and this is the most favorable time in which to operate.

If, however, the suppuration continues this epithelium disappears and the alveoli become obliterated and there is found a true connective tissue layer composed of the fused alveolar walls observed in chronic abscesses. A condition most unfavorable for a complete cure and one which favors the establishment of a permanent bronchial fistula.

The problem is to give each patient a full chance to reach this more favorable stage and at the same time not to allow the disease either to extend indefinitely or to drift into the chronic abscess with thick fibrous walls which is unfavorable for radical cure by operation or any other method of treatment.

This can only be done by the close coöperation of physician and surgeon in the study of the case, and particularly the comparison of frequent x-ray plates of the chest.

From three to four weeks of such medical treatment may usually safely be employed to determine the necessity for operation, in which time many will recover without it.

More radical operation in the form of lobectomy, although successful in one of the three cases in which it was attempted in this series, must still be considered as an extremely hazardous procedure, although theoretically it might be considered the ideal treatment when by an improvement in surgical technique it is possible to diminish the present great operative risk. The successful case was one seen by Dr. Miller. The lobectomy was performed by Dr. Lilienthal who obtained a truly brilliant result.

Artificial pneumothorax is being quite extensively advocated for the treatment of acute pulmonary abscess. Our experience has been

very limited, it being used only in the case of seven patients, none of whom improved and two of whom died.

We do not wish, however, to have this considered as our final judgment upon this method of treatment, for it seems quite possible that it may be of help in acute abscesses freely opening into a bronchus and situated near the hilum of the lung.

We do wish to emphasize, however, that its use in peripheral abscesses is attended with great danger of rupture of the lung with subsequent pyopneumothorax, and this actually happened in two of our cases and has been quite frequently reported in the literature. Also it is our experience that this method of treatment may do harm in the case of abscesses not situated near the hilum by exerting pressure on the proximal portion of the bronchus and thus shutting off drainage, and it also may inhibit drainage by preventing the normal expulsive power of coughing. In one case we employed this method very successfully for the control of large and repeated hemorrhage.

Bronchoscopic Lavage.

Bronchoscopic suction and lavage has also been very favorably advocated in the treatment of pulmonary abscess. Our experience has been limited to three cases which were referred to the laryngoscopical division of the hospital for this treatment. Of these three cases which were referred to the laryngoscopical division of the hospital for this treatment. Of these three cases, one was improved, one not improved, and one died, apparently from a rupture of the lung directly following the treatment. We have no desire to record a final judgment upon this method from our scant experience, and bronchoscopic examination is highly to be recommended in cases of suspected foreign body in the lung and for the diagnosis.

We are, however, rather deeply impressed with the fact that this procedure is a very trying ordeal for acutely ill patients, and also we are not convinced that it is unattended by a real risk to the patient.

Analysis of Cases.

Sex. Forty-five cases were in men and fifteen in women, a proportion of three to one in favor of men. This is in accord with the reports of other observers.

Etiology. The cause of pulmonary abscess in a given case is not always easy to establish. As far as we could determine from the histories the etiological factors in this series may be grouped as follows:

No definite cause . . . . (Primary abscess)	22
Secondary to pneumonia	16
Secondary to post-operative pneumonia . . .	3
Secondary to tonsilectomy	8
Secondary to other operations	5
Following tooth extraction	2
Following submersion	2
Following foreign body inhalation	1
Following trauma to chest	1
	60

The high incidence of cases (36.6% of all) to which no definite primary cause may be ascribed is noteworthy. Also the occurrence of abscess following tonsillectomy is lower than in many other series reported.

Duration of Disease.

The duration of the disease before coming under observation is important from the standpoint of methods and results of treatment.

Less than one month	23
From one to three months	23
From three to six months	7
Over six months	7

Forty-six of these cases (76.6%) may be therefore considered as acute abscesses.

The results of treatment show that of the twenty-three cases of less than one month duration fourteen (60.8%) were cured and five died, a mortality of 21.3%.

Of the twenty-three cases from one to three months six (26%) were cured and nine (39%) died.

Of the seven cases of three to six months duration two (28.5%) were cured and three (42.8%) died.

Of the seven cases lasting more than six months three (42.8%) were cured and three (42.8%) died.

The generally recognized predominant importance of early treatment is again emphasized.

Location of the Lesion.

The lesions were in the right lung in forty cases, in the left lung in nineteen, and in both lungs in two cases. They were in the lower lobe in forty cases, in the upper in nineteen cases, in the right middle lobe in one case, and in both upper and lower lobes in one case. Three cases had multiple lobe involvement.

Of the nineteen upper lobe cases ten (52.6%) were cured and six (31.5%) died. Of the forty lower lobe cases sixteen (40%) were cured and twelve (30%) died.

These results indicate a somewhat more favorable prognosis in upper lobe cases, but not so marked as some other series have shown.

Results of Treatment.

As has already been described, our experience led us to modify our methods of treating these cases very radically.

We are therefore reporting the results in one series as a whole and in addition are reporting separately the comparative results obtained by the two radically different methods. These results are indicated by an early series of twenty-nine cases in which early radical surgery predominated and second later series of thirty-one cases in which medical treatment was almost invariably first tried with postural drainage as the most important feature.

Results of Complete Series. 60 cases.

Cured 26. Improved 6. Unimproved 8. Died 20.

A mortality of 33.3%.

Results of Early Series. 29 cases.

Cured 11. Improved 2. Not improved 4. Died 12.

A mortality of 41.4%.

Results of Later Series. 31 cases.

Cured 14. Improved 5. Not improved 4. Died 8.

A mortality of 26%.

The comparison not only shows the later series to much better advantage in increased cures and diminished mortality, but also indicates a larger number who were improved, although not cured, the combined cured and improved cases being 45% in the early series

and 61% in the later one. The fact that by medical treatment a certain number of patients were able to go on fairly comfortably without constitutional symptoms and with alleviation of disturbing cough and expectoration influenced not a few to decline advice to undergo an operation advocated in the hope of effecting a radical cure.

Results Obtained by Various Methods of Treatment.

In comparing the two major therapeutic procedures which we have employed in this series, we find that postural drainage was used in thirty cases of which fourteen (46.6%) were cured, four (13.3%) were improved, eleven (36.6%) were unimproved and one (3.3%) died.

In the operative cases, of which there were twenty-seven in all, eleven (40.7%) were cured, one (3.7%) was improved, and fifteen (55.6%) died.

The very much higher mortality in the operative cases is very striking, although it must be borne in mind that there are here included eight cases in which medical treatment had first been tried and had failed.

It also must be noted that the operative results in the later series, in which we had arrived at a more satisfactory method of selection of cases as regard the time most suitable for operation, are much more favorable than in the early series. In fourteen operative cases of the early series the mortality was 71.5%, while in thirteen cases of the later series it was only 38.5%.

Summary.

(1) Acute pulmonary abscess is a comparatively common and extremely serious disease.

(2) Great opportunity for improvement in methods of treatment exists. This can best be met by close combined study of each case by physician and surgeon.

(3) It seems probable that many cases are abscesses from the start without going through the intermediate stage of pneumonia.

(4) Our studies of the bacteriology would indicate that anærobic micro-organisms may play an important pathogenic rôle in their causation. Further study of these organisms seems important.

(5) The treatment of acute abscess is primarily a medical problem. When surgery is necessary, results are far more successful after preliminary medical observation and treatment.

DISCUSSION.

PRESIDENT: I was very much interested in the paper presented by Dr. Miller which throws a new light on the abscess of the lung. At the meeting of the American Laryngological Association in Atlantic City, Dr. Fetroff of Philadelphia was awarded the Casselberry prize for his thesis on abscess of the lung, its etiology and treatment. I, unfortunately, reached the meeting too late to hear the paper, but in talking to Dr. Tetroff later, I obtained his ideas with regard to the method which he described as the cause of abscess of the lung. These were the results of experiments upon animals. His view is that abscesses are much more the result of septic infarct than from aspiration. This view is entirely opposite, in a certain way to our practical experience. In my earlier paper on the subject I was rather inclined to believe that these abscesses were of septic infarct rather than aspiration; but after practically applying a thorough asepsis of the buccal cavity and teeth, the use of Trendelenburg's position, and other measures, I have come to the conclusion that the majority of cases arise from aspiration. For this reason I have been employing this method of surgical interference. preventing my assistants and internes from unduly swabbing.

Practical experience seems to be more valuable than theoretical research in these cases, and since in our hospitals we have been carrying out aseptic procedures, we have had hardly any abscesses of the lungs. Abscess of the lungs that have lately come under our observation are due to the internasal work, wherein the infection necessarily must be from aspiration.

As to the treatment by pneumothorax, I cannot agree with the observations along that line in the paper, or Dr. Price. Our experience has been quite large and we find artificial pneumothorax is the more valuable method to be employed in these cases.

Only two months ago I was called in consultation in a case that had been operated on a week before. It was a well located abscess in the right lung and about the medium position. Artificial pneumothorax was applied and the patient improved from the first injection into the pleural cavity. Each time it was applied there was a marked improvement, until the third treatment the patient was practically well. In two weeks she left the hospital. I saw her a few days before I came and she was absolutely well. I could go on and enumerate case after case of this type. Very rarely do we have rupture of the abscess in the treatment through pneumothorax. A thorough study of the situation, the age of the abscess, the presence of adhesions should prevent it from occurring. No chronic cases should have pneumothorax. I believe that if artificial pneumothorax is applied in the acute cases early, especially where the

abscess is deeply located the result is almost certain. Abscesses in the upper lobes are decidedly more favorable, they drain normally and evacuate themselves. I have seen several cases of this kind who after a month have drained perfectly and have recovered.

Dr. Nichols: How large an inflation did you use? I have had three cases of lung abscess during the last year with which I used pneumothorax treatment without success.

President: 100 to 150 cc. every three or four days. Dr. Tewksbury uses a large amount of air pressure.

The reason the antrum infection causes the infection in the lower tract is self-evident to most of you, if you remember that the drainage of the antrum is most active when the patient is in a recumbent position, that is in sleeping, and when he is less able to protect himself. The other sinuses drain when the patient is in an erect position, or when they are awake and able to protect themselves.

Dr. Otis: The last three cases of abscess of the lung which I saw who were operated upon all had a fatal termination and all were acute. One must confess that the surgical handling of a lung abscess is dangerous. I recall a case in the influenza epidemic of 1918 when a lung abscess emptied itself through a bronchus and completely cleared up in a short time. Chronic lung abscesses in my experience offer a very unfavorable prognosis. They drag on indefinitely and finally result fatally. Here is a field for thoracic surgery which has already achieved some notable triumphs. Abscesses resulting from tonsillectomy, of which there are apparently not a few, as has been reported, I think, by our president, Dr. Richardson, have been successfully treated by artificial pneumothorax, and it would seem that this procedure should be one of choice before surgical interference should be resorted to. Only after postural drainage, bronchoscopy and artificial pneumothorax have been tried should surgery be resorted to. This, I believe, is the course recommended by thoracic surgeons.

Dr. Dunham: I would like in the discussion to have Dr. Miller explain a little more carefully just when and how that rupture took place, so that we may stop this practice, or do it more carefully.

Dr. Elliott: I have treated four cases in which the abscess was close to the pleural surface, and in none of these has there been any rupture. In some of these there have been adhesions so only a partial pneumothorax could be induced. One case in which there were no adhesions did rupture into the pleural cavity but recovered after resection.

Dr. Claytor: My last experience with the production of artificial pneumothorax in lung abscess was not encouraging. In this case there was an abscess which appeared to be in the middle of the lower lobe of the right lung. Bearing in mind Dr. Tewksbury's favorable report on this method of treatment I got him to inject air in this case. There were four injections at weekly intervals beginning at 150 c.c. and increasing to 400 c.c.

There was a decided improvement after each of the first three treatments

(lowering of temperature, lessening of cough, etc.) but three days after the fourth there was a rise of temperature to above 103°; severe pain in the right lower quadrant; rigidity and tenderness; a leucocytosis of 20,000.

A surgeon made a diagnosis of probable appendicitis but when I saw the patient two days later the pain was more diffused over the right lower chest and right abdomen and the presence of pleural friction convinced me that the symptoms were produced by an acute pleurisy. The signs of air and fluid in the chest rapidly developed.

An incision was made and three pints of pus were drawn off, but death resulted a few days later. Autopsy showed a small abscess cavity in the lower lobe which had ruptured into the pleural cavity in the diaphragmatic region.

One is forced to the conclusion that artificial pneumothorax is not without its dangers in these cases of lung abscess.

Dr. Packard: Did Dr. Miller treat by postural treatment the old abscess cases? How does he treat by postural method? How long at each time and how often?

Dr. Elliott: I have treated four cases in which the abscess was close to the pleural surface, and in but one of these has there been any rupture. In some of these there have been adhesions so only a partial pnumothorax could be induced. One case in which there were no adhesions did develop a rupture in the pleural cavity. I have seen at least three or four cases during the same period where the rupture occurred without pneumothorax, and I wonder if Dr. Miller is satisfied that there is more danger where pneumothorax is.

Dr. Price: I have treated four cases of lung abscess by pneumothorax with no success. Didn't have any bad luck, but no good luck. I have come to the belief that operation was the best thing for old abscess cases. I had a patient who had a lung abscess for some time. The abscess was on the top of the right lung. It was drained very successfully. In the meantime she has developed an abscess in the left lung. This was drained and found to be in a worse condition than in the right lung.

Dr. Miller: I think it is interesting to discuss this subject this year, and the medical profession owes a great deal to our president for having brought to the attention of the profession a number of cases that shows the dangers of operation on the air passages to the lungs. Preventive work is really more valuable than anything else as far as the question of treatment is concerned.

Our method is this: Try posture drainage first. It does not take more than a week. It is done by really inverting the patient, preferably toward the opposite side. They will begin to invert themselves with difficulty and then you increase it gradually. In the course of two or three days they can stay there until they have been drained. They are comfortable for four or five hours. In the meantime they have little or no cough. This brings down their temperature. This is the method of preference at first, but all of the time keep watching the case daily.

We have not closed our eyes to the possibilities of pneumothorax. Here

and there are cases in which it would seem for some reason or another that they do rupture, and it seems to me that they do rupture as a direct result of pneumothorax. I am interested in what Dr. Dunham says that it does help. If you do it very carefully it may be done. In cases where the abscess is near the surface you must bear in mind there is some risk. As Dr. Tewksbury says, the cases must be acute. They do better when they are near the root of the lung. Do postural drainage first and then artificial pneumothorax. Nothing to be lost by postural drainage. There is need for very much careful analysis of the type of cases which want the various kinds of treatment. All methods of treatment are valuable but there are certain times to use them. Try for a short time with your postural drainage first. In a week or two you can tell. Then select another measure. You will find that postural drainage is a less risky procedure. In cases of peripheral abscess, they should be handled with great care, so far as pneumothorax is concerned.

AN X-RAY STUDY OF APPROXIMATELY FIFTEEN HUNDRED CHILDREN BEFORE AND AFTER TONSILLECTOMY UNDER ETHER.

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Two years ago a great deal of interest was aroused in regard to diseased tonsils among the children of Rochester. All of the children of school age (the majority from six to twelve years of age) were examined by school physicians and a group suitable for operation was referred to the Tonsil Clinic. Seven thousand, eight hundred and seventy-two from this group were selected to be operated upon. To our minds this afforded an ideal opportunity for studying the chests of apparently normal children with the x-ray.

Our purpose in this study was threefold. First, two determine just what was a normal chest for a child; second, to note if the removal of the tonsils caused any change in the x-ray plate of the lungs; third, since in the illness of so many tuberculous patients, the history dated back to an operation under ether, to ascertain whether or not ether caused any noticeable changes in the lungs as regards the x-ray.

During March and April, 1921, we x-rayed 2,226 children. These x-rays were taken the day before the tonsillectomy. In July and August we repeated about 1,200 children at the various schools. Again in November and December 1,000 children were x-rayed. Out of these 2,226 children there were two exposures on 1,522 and three exposures on 550. We are basing our results only on those children who had two or three plates taken. Our findings agree with those of the committee appointed by the National Tuberculosis Association (Am. Jr. of Roent. 9, 537.543, Sept. 1922), in that they are largely negative. On the other hand some of these children showed marked changes in their chest plates following tonsillectomy under ether. One such case was E. D., girl, age 7, who developed pulmonary tuberculosis following her operation. Her plates taken April 1921,

showed increased linear markings and beading throughout both lungs. She was admitted to the sanatorium in January, 1922, with the history of a "cold" following tonsillectomy, loss of weight and loss of appetite. There were a few inconstant rales at the right base anteriorly, and D'Espigne's sign. The x-ray plate showed no change on the right, but on the left a markedly increased hilum, and a dense area in the second interspace with connecting lines to the hilum. Another plate taken in April, 1923, shows the area described above not as dense after fifteen months of sanatorium treatment. This girl has gained thirty-five pounds, and has improved in every way.

J. G., boy, aged 6, showed a chest practically normal before operation. On a plate taken a year later there was definite beading above and below the right hilum, while the hilum shadow on the left was increased.

Another child who showed a spread after tonsillectomy under ether was A. F., boy, aged 7. On the plate taken before tonsillectomy the right hilum was slightly increased, and there was a narrow band extending out into the right lung which suggested interlobar thickening. Several months after the operation the child's plate showed a dense shadow four or five cm. in diameter and the same dense band extending out from the hilum.

M. I., girl, aged 8, showed some beading in all directions from the hilum of each lung. In a plate taken nine months after the operation there is an increase in the size of each hilum, and a more definite beading throughout each lung.

F. D., girl, aged 11, showed on the left a dense area at the apex and a mottled appearance in the first interspace, while on the right there was a small dense area toward the apex. A diagnosis of pulmonary tuberculosis was made from the plate. We advised against operation, but through a mistake the operation was done. In November, 1921, there were no physical signs except diminished breathing over both uppers. On reëxamination in September, 1922, there were dullness and increased whispered voice to the third rib on the right, and large moist rales over the upper half of the left lung. At this time her sputum was positive. This case illustrates the clinical observation that many patients date their illness with pulmonary tuberculosis back to an operation under ether. Is it not possible that

the irritation from the ether has re-activated a quiescent tuberculosis?

We do not believe that all the changes found were due primarily to ether. In fact, some of the children not operated upon showed changes just as definite. A boy, F. P., aged 5, was x-rayed in March, 1921. The plate was practically clear. A plate taken in June, 1921, showed the right hilum enlarged, with increased linear markings in all directions from the hilum. This boy was admitted to the sanatorium in February, 1922. At this time his physical findings were bronchial breathing, increased whispered voice, and rales to the third rib, right front; and rales from apex to the base, right back. His x-ray on admission showed dense infiltration upper two-thirds on the right, and on the left increased linear markings upward from the hilum. In April, 1922, one c.c. of sputum was injected into a guinea pig. At the autopsy on the guinea pig many hard tubercles were found at the point of injection. Smears were made which showed tubercle bacilli.

In April, 1923, this child developed three cold abscesses—two at the level of the sixth rib near the posterior axillary line on the right side, and one at a corresponding area on the left side. A plate taken in April, 1923, showed on the right a dense shadow extending from the clavicle to the base. During the past year this child's general condition has shown marked improvement. This improvement has also been noted in his x-ray plates.

Among other chest conditions that came under our observation were several cases of lung abscess. The first was that of A. L., boy, aged 13, who was operated upon in March, 1921. His plate before operation was practically normal. He was admitted to the General Hospital two months later on account of cough, pains in left chest, and temperature 100.2 to 102 degrees. Over the upper left chest there were dullness and diminished breath sounds, but no rales. Another plate taken in July, 1921, showed a large dense area in the middle third of the left lung—probably lung abscess. In a plate taken in August there was the same dense area described above in the left lung, with an area of lesser density in the center—probably due to a cavity. He was in the sanatorium from November, 1921 to May, 1922. During this time he received general sanatorium treat-

ment, and in addition he was given postural drainage. He improved rapidly, lost his symptoms, and gained ten pounds. In his plate taken in January, 1922, the shadow of the lung abscess has almost disappeared. His condition on discharge was excellent.

Another lung abscess was that of F. D., girl, aged 12, whose x-ray plate was practically normal before tonsillectomy under ether. She was admitted to the General Hospital in April, 1921, with a history of tonsillectomy fifteen days previous to admission. She had temperature of 101 degrees, and had had cough and expectoration for seven days. On physical examination there was dullness and diminished breath sounds over right base posteriorly. Her x-ray plate showed a dense area in the first and second interspaces of the right lung. In a plate taken two weeks later this dense area was larger, and in the center there was an area of lesser density which was probably due to cavity. After leaving the hospital her cough persisted and she was sent to the sanatorium. At this time (March, 1922), there were a few fine rales at the right apex which persisted over a period of several months. The x-ray in June, 1922, showed increased linear markings upward and outward from the hilum, right side. This case was particularly interesting on account of the fact that these rales may have been due to incipient tuberculosis or to the lung abscess. After having this patient under observation for seven months she was discharged as doubtful tuberculosis. Her general condition was excellent; she had gained six pounds and lost all symptoms.

According to Wessler (Am. Jr. Roent. 6:161, April, 1919), lung abscesses following aspiration usually occur in the upper part of the lung, and frequently heal without any treatment. Two of our cases were in the upper third of the lung, and the other two were in the middle third. The two in the upper part healed, and while the two in the middle third have shown some improvement, there are still "markings" in the lung.

There were many interesting chest conditions shown in the series of plates that were taken. Briefly, some of these were as follows:

M. M., girl, aged 11, on her plate before operation was practically normal, but after operation there was a dense area in the middle third of the left lung. At the end of one year most of this

area had clared up, leaving a narrow dense band extending out from the left hilum. Diagnosis: Lung abscess.

S. M., boy, aged 11, had a dense area in the middle third of left lung 8 or 10 cm. in diameter—before operation. Three months after operation there was very little change in this shadow. In a plate taken two years after operation most of this dense area had cleared, but there were still a few markings in the third interspace. We feel that this was a lung abscess, but there was some doubt as it may have been due to an unresolved pneumonia or tuberculosis.

L. D., boy, aged 8, in his plate before operation had a large dense area six or eight cm. in diameter extending out from the right hilum. This shadow was probably due to pneumonia or hilum tuberculosis. Although an effort was made, no plate could be obtained after the operation, so that neither diagnosis could be substantiated.

T. D., boy, aged 7, showed a definite enlargement in the region of the left hilum four months after operation. This plate suggests hilum tuberculosis.

R. P., girl, aged 13, on a plate taken eight months after operation showed a slightly increased shadow of the left hilum, and a very definite increase at the lower pole of the right hilum.

H. N., girl, aged 12, on a plate taken before operation showed a small dense trianugular lesion under the third rib toward axilla, right side, with a faint line connecting the lesion with the hilum. The lesion was probably pulmonary tuberculosis. In a plate taken nine months later the chest was clear.

M. C., boy, aged 7, showed a dense area above the right hilum extending up to the clavicle. In a plate taken a year after the operation this area was almost clear. This plate suggests hilum tuberculosis.

L. G., girl, aged 9, showed a definitely increased hilum shadow on the left side, on comparing plates taken before and after the operation.

A. B., boy, aged 8, showed a definite beading throughout both lungs. There were many plates of this type. We did not feel that a diagnosis of pulmonary tuberculosis could be made on this. These changes in the lung may have been due to some previous disease, as measles, or whooping cough, which most of these children had had.

W. D., boy, aged 9, before operation showed a calcified ring at the left hilum. On plates taken one month and ten months after the operation this same calcified area was seen.

A. K., boy, aged 8, showed many calcified areas at each hilum —especially marked on the right.

E. M., boy, aged 10, showed calcified areas at the left hilum.

R. M., girl, aged 9, showed large calcified areas at each hilum—much more definite on the right.

M. W., girl, aged 12, showed many large calcified areas at each hilum—more definite on the left.

R. H., boy, aged 6, on the plate before operation showed a calcified area out from the right hilum toward the axilla. There was a narrow dense band connecting this area with the hilum. Three months after the operation the calcified area could be seen more definitely, but the band connecting this with the hilum was not as dense as before.

SUMMARY OF X-RAY FINDINGS.

Pulmonary tuberculosis	26	1.7%
Hilum tuberculosis	13	.8%
Pneumonia	3	.2%
Lung abscess	4	.3%
No gross lesion	1476	97.0%
Total	1522	

At the end of one year one hundred and forty-two of these children were picked at random and examined by the staff from the sanatorium, with results as follows:

Age				Sex	
1-4	5-9	10-14	15-19	Male	Female
6	69	61	6	76	66

Family History—Negative tuberculosis 139

Positive tuberculosis 3

Previous Diseases: Measles 89, whooping cough 55, chicken-pox 46, mumps 20, diphtheria 9, pneumonia 13, influenza 30, bronchitis 10.

Symptoms: Cough 16, expectoration 2, pain in chest 5, shortness of breath 5, lassitude 13, poor appetite 7.

X-ray Diagnosis: Positive tuberculosis 3, suspicious tuberculosis 2, negative tuberculosis 136, lung abscess 1.

Diagnosis from Symptoms, Signs and X-Ray Findings: Positive tuberculosis 3, suspicious tuberculosis 1, negative tuberculosis 137, lung abscess 1.

PHYSICAL SIGNS.

Positive physical signs	31	Rales upper half	5
Negative physical signs	111	Rales lower half	9
		D'Espinge's	18
Total	142	Squeaks	5
		Dullness at apex	1
		W. V. at apex	2

It is almost needless to say that we have not established a normal chest plate for a child. Apparently there were no changes in the plates caused by the removal of the tonsils. In several children with tuberculosis there was a spread after tonsilelctomy under ether. On the other hand, several children diagnosed as tuberculous seemed to improve after operation. Most of the children who were apparently normal before operation showed no change afterward. To prove the effect of ether on pulmonary tuberculosis, it would seem necessary to study two groups of patients with pulmonary tuberculosis. One group should be operated upon under ether, and the other group should be operated upon under gas oxygen. If these were major operations rather than tonsillectomy, the knowledge gained would be more valuable.

In addition to the positive findings there were a great many plates which showed increased linear markings and beading; and a great many which seemed to show nothing abnormal. So, from a single plate, or from a series of plates, it is impossible to foretell whether or not the child will develop pulmonary tuberculosis after a few years. There are other things of much greater importance, such as: the health habits of the child, the food, and the home conditions.

CHRONIC BRONCHITIS.

By JAMES B. HAWES, 2nd, M.D.,

BOSTON, MASS.

Richard Cabot once told me that he doubted if there was any such thing as chronic bronchitis. He gave as his reasons for this statement that during the twenty years he had conducted clinico-pathological exercises at the Massachusetts General Hospital no case of chronic bronchitis had ever come to autopsy. In his opinion, chronic bronchitis was the name given to a group of symptoms caused by:

(a) A weak heart with congestion or œdema.

(2) Bronchiectasis.

(c) Interstitial pneumonitis.

I am unwilling to admit that because patients do not die of a given condition or because it is so located that an autopsy cannot be performed or because it cannot be demonstrated by x-ray (which would be disputed by many roentgenologists) that such a condition does not and cannot exist. I do freely admit, however, that chronic bronchitis like "general debility," "anemia" and "run-down" is a diagnosis which in far too many instances covers ignorance and a lack of thoroughness. On the other hand, I would stoutly maintain that just as there is an acute bronchitis, rhinitis and coryza so there is a sub-acute and chronic bronchitis and that this condition is not necessarily an undiagnosed bronchiectasis or the result of an unnoticed or underestimated cardiac weakness. Were I asked whether chronic bronchitis was ever a primary condition I should state most emphatically that cases of primary chronic bronchitis are rare.

There is much literature on this subject. Osler (1901 edition) considers chronic bronchitis a definite clinical entity and even speaks of its pathology with a special reference to the condition of the mucous membrane found in such cases. In the 1907 edition he says

it may follow repeated attacks of acute bronchitis. He makes a special mention in each edition of a form of bronchitis with which I am not familiar, found in women between the ages of 20-30 years which may continue indefinitely without serious impairment to their health.

Lord speaks of chronic bronchitis as a symptom rather than as an independent condition.

Babcock devotes an entire chapter to this subject, evidently regarding it as a definite clinical entity. Instead of considering the chronic bronchitis as secondary to cardiac weakness and arterial and circulatory changes he believes that the cardiac lesions are secondary to the bronchitis and its accompanying emphysema.

Landis and Norris consider primary chronic bronchitis a rare condition and speak of "some who refuse to recognize the existence of a primary chronic bronchitis under any circumstances." Primary cases, in their opinion, comprise those due to exposure to dust such as found in millers, stone-cutters, coal miners, etc. "Cotton fever"—that condition often met with in young men who first enter the cotton business—is probably a chronic bronchitis coming in this category.

It is needless to quote further literature on the subject. It is evident from the references that I have given that chronic bronchitis is considered by many eminent authorities as a definite clinical entity but that also there is considerable difference of opinion on the subject. The various men with whom I have discussed the subject of chronic bronchitis have as many different opinions concerning it, its etiology and classification.

Dr. Charles L. Minor rather doubts if there is such a thing as chronic bronchitis because he does not often see it although he would not absolutely deny its existence. As he says, if he lived in New England he doubtless would meet it oftener than in the south.

Dr. W. Jarvis Barlow believes that chronic bronchitis does exist but that it is always a secondary affair.

Dr. Henry Christian believes that it is a definite clinical entity. He calls attention to the interesting combination so frequently met—a chronic bronchitis with emphysema.

Dr. H. R. M. Landis believes that the only primary bronchitis that he would recognize would be that due to the inhalation of inorganic dust such as in pneumoconiosis; all other instances of

so-called chronic bronchitis are secondary to other changes. He believes that as a general rule it would be a good thing if the general practitioner were to forget chronic bronchitis or at least to bear in mind that it is commonly the outstanding feature of a more serious ailment.

The classification of chronic bronchitis which I use is a purely clinical one, as follows:

1. Primary chronic bronchitis. In this group I would include —(a) Cases where the bronchitis is due to some irritating substances such as cotton, flour or some other form of dust. (b) Gouty bronchitis. This is a chronic cough occurring in persons of middle age, or past middle age, who are fat, plethoric, usually with a high blood pressure and other symptoms of insufficient elimination.

2. Secondary chronic bronchitis, including — (a) Cases in which the bronchitis is secondary to an infection in the upper respiratory tract such as infected tonsils, sinuses, adenoids, carious teeth, etc. (b) Chronic bronchitis following some acute pulmonary infection such as acute bronchitis, influenza, broncho-pneumonia, etc. (c) Bronchitis following gassing in ex-service men.

3. Wrong diagnoses. A large group of cases with a chronic cough and with or without sputum which would fall in Richard Cabot's category of mistaken diagnosis, where the cough and other symptoms are due either to a weak heart as in many elderly persons, particularly in New England, or an unrecognized bronchiectasis.

The signs and symptoms of chronic bronchitis need only be mentioned briefly:

(1) *Cough,* usually with considerable sputum but often dry, paroxysmal and irritating, resembling whooping cough, usually worse in the winter months and especially in damp weather. Many men who were gassed are veritable human barometers and can always tell when a storm is brewing.

(2) *Sputum.* This may be abundant, often it is purely mucoid, but usually muco-purulent and occasionally consisting of almost pure pus. Organisms of all sorts may be found. Bloody mucous is frequent; actual hemorrhages of one teaspoonful or more undoubtedly do occur but are rare.

(3) *Constitutional signs and symptoms.* Loss of weight, fever,

and rapid pulse are not common. Shortness of breath, weakness and ease of tire, especially in gassed ex-service men, are often extreme. In many instances these symptoms in ex-service men at least are more often due to neurasthenia or a neurosis of some sort rather than to any bronchitis which may exist.

(4) *Signs in the lungs* may be lacking or nearly so or there may be rales of all kinds, wet and dry, coarse and fine, sibilant, sonorous, etc., usually located at the bases, usually bilateral and often associated with harsh, prolonged expiration and other signs of emphysema. Dullness and other voice and breath changes are usually lacking.

Now let us consider more in detail the various group of cases as classified above.

1. Primary chronic bronchitis.

(a) There is very little to be said concerning the first sub-heading of this group,—chronic bronchitis due to some form of dust or iritating substance. Treatment naturally consists of the removal of the cause.

(b) Gouty bronchitis. This form of primary bronchitis is to me of distinct interest although some of my friends doubt its existence. This condition usually occurs in men and women of past middle age. There is usually persistent cough which continues month after month regardless of wind or weather. To use automobile parlance, "their cylinders are carbonized and there is sand in their carburetors." They are apt to be red-faced, plethoric, overweight, with an increased systolic blood pressure, often with albumen in the urine. The cough is hard and dry; rales, usually dry squeaks and wheezes, are present at both bases often accompanied by signs of emphysema. X-ray examination often shows a somewhat enlarged heart along with what roentgenologists call peribronchial thickening.

Treatment consists of depletion and elimination. The total amount of food taken should be reduced at least one-third. Meat should be prohibited except once or twice a week and salt, condiments and meat extractives should be reduced to the minimum. The patient should drink 10-12 glasses of water daily and should keep the bowels open by means of a saline laxative. Iodide of potash taken regularly for the first two or three weeks, then every other week until

finally it is taken one week out of every month, is of distinct benefit. Oil sprays or, occasionally, steam inhalations, are of great help in alleviating the paroxysms of cough. Exercise such as walking, riding or golf is a most important factor. With this type of bronchitis, as indeed for most cases of chronic bronchitis, cold air at night should be avoided and the thermometer in the bedroom should not be allow to go below 45°, or better still, 50°. These patients will often show a wonderful improvement on such a regime as this.

2. Secondary chronic bronchitis.

(a) The first group consists of cases in which the bronchitis is due to an infection higher up. Signs in the lungs are usually as above mentioned with the addition of a few cases where signs such as dullness, rales, etc. closely simulating an early tuberculosis are present at one of both apices. French observers have done a real service in calling attention to this group which still needs further investigation and study, however. Treatment of these patients naturally consists in removing the source of infection higher up. All other treatment is purely temporary and palliative.

(b) Chronic bronchitis following some acute pulmonary infection such as influenza. I am one of those who firmly believe that just as there is post-influenzal depression of which I have had a vivid personal experience there is also a post-influenzal bronchitis.

Such patients are apt to be in poor physical condition. Most of them have taken cough mixtures of all sorts and varieties and are generally discouraged and disgusted with life. What they need most of all is encouragement, iron, rest and particularly a change. By sending them away for a time even if only a short distance where climate cannot possibly be a factor but where business and housekeeping cares are left behind and where they can rest mentally as well as physically, wonderful improvement often takes place in a surprisingly brief period.

(c) Chronic bronchitis following gassing. I have never seen any acute gas cases but I have seen hundreds of men who at least attribute their present symptoms—cough, sputum, often bloody, and associated with frank hemorrhages, pain, constriction in the chest, shortness of breath and general weakness—to the effects of the gas. I quite agree with Dr. Cabot in his suggestion that the pathology of

many of these gas cases may be an interstitial pneumonitis and occasionally a bronchiectasis. In the great majority, however, bronchiectasis can be definitely ruled out. As far as interstitial pneumonitis is concerned, I and the x-ray men with whom I have talked at first were convinced that there was a definite increase of fibrous tissue in the lungs of men who were badly gassed. Recently I have come to doubt this so that I admit freely, and I believe roentgenologists will agree with me, that I do not know what pathological changes gas causes in the lungs. Often there is considerable peribronchial thickening, especially toward the bases, distinctly more than normal, but I am unwilling to call this an interstitial pneumonitis.

These gassed men, as everyone knows who has dealt with them, are extremely difficult propositions. Most of them at one time or another have been labelled as "Tb. suspects" or actual consumptives which has certainly done them no good. At least they can be relieved of this burden. Many are nervous and apprehensive and need a frank, encouraging talk with words of hope and cheer more than anything else; many of them need a severe and even brutal talking-to and to be urged to go to work. They are often helped by doses of iodide of potash, occasionally with addition of belladonna, if expectoration is profuse. Large amounts of water up to 8-10 glasses daily and free movements of the bowels should be insisted upon. Wind and dust and cold air at night are especially harmful. With many of these men, as with other cases of chronic bronchitis, coughing has become a habit which must be broken up. In this bromides are often of great value providing it is carefully explained to the patient what the medicine is for and that it is purely a temporary measure.

3. Wrong diagnoses. This last group which is not bronchitis at all includes those patients with a chronic cough due to some other cause, such as a weak heart, bronchiectasis, interstitial pneumonitis, or others such as an undiscovered tuberculosis, syphilis, malignant disease, asthma, enlarged bronchial gland or a multitude of other causes. Treatment consists in finding the cause, its removal if possible, and if not, instituting proper treatment.

I have not included asthmatic bronchitis in this discussion. This interesting condition is too closely allied to true bronchial asthma

to be classified as a type of chronic bronchitis. Likewise, in the treatment of chronic bronchitis I have made no mention of the use of vaccines. In my opinion, they are rarely, if ever, of value. In far too many instances the physician at once prepares an autogenous vaccine or more often uses a commercial preparation and gives the patient a series of injections instead of trying to trace down the bronchitis to its source and removing the cause. I regard the use of vaccines in chronic bronchitis as distinctly a last resort measure.

I would summarize my ideas on this subject as follows:

I. I believe that chronic bronchitis is a definite clinical entity, occasionally primary but usually secondary to something else.

II. Congestion following a weak heart, bronchiectasis, chronic interstitial pneumonitis are often mistakenly called chronic bronchitis.

III. Chronic bronchitis may be classified as follows:

1. Primary Chronic Bronchitis. Due to—(a) Dust or other iritating substances. (b) Gouty bronchitis.

2. Secondary Chronic Bronchitis. Due to—(a) Chronic infection of the upper respiratory tract. (b) Acute pulmonary infection. (c) Gassing.

3. Mistaken Diagnoses.

IV. Treatment depends on finding the cause, removing it if possible, or giving proper treatment.

V. Asthmatic bronchitis is a form of asthma and not a true chronic bronchitis.

VI. Vaccine treatment should be looked upon as a last resort measure. It should never supplant or replace other therapeutic measures and should be only used after a detailed search for other causes of chronic cough has been made without avail. The chief requisite in the use of vaccines is to see that they do no harm.

DISCUSSION.

DR. BABCOCK: I am not certain whether a chronic bronchitis may in the strict sense exist without associated small bronchiectasis or not, particularly when the physical findings are limited to the base of one or both lungs. In a strict pathological sense the condition formerly termed capillary bronchitis is probably associated with if not dependent for its chronicity upon small areas of broncho-pneumonia. Changes in the walls of the bronchioles undoubtedly

are present and overshadow any other condition, and so we may designate the recurring or persistent cough and the fine mucous rales as a chronic bronchitis. Furthermore, I believe that definite changes sometimes take place in the larger bronchi which do really constitute a chronic bronchitis.

Dr. Otis: Any dispensary physician who is seeing a large number of pulmonary cases must agree, I think, with Dr. Hawes that there is such a disease as chronic bronchitis. At least I do not see how he can interpret otherwise the symptoms and physical signs which we usually attribute to this disease. Undoubtedly there also exists at times some bronchiectasis or possibly a patch of broncho-pneumonia which we include under the general head of chronic bronchitis, but it is often difficult or impossible to make the differentiation.

Dr. Webb: We were very much surprised in the service men to find so many cases of bronchitis. We wrote to Dr. Bushnell and he replied that it was due to the high food that they were getting. Much of the bronchitis left after we cut down the diet and exercised.

Dr. Pryor: Discussing the paper by Dr. Hawes, allow me to state very briefly that probably the most important test to be employed in distinguishing between chronic bronchitis and bronchiectasis is to tip the patient upside down. The procedure often helps us strikingly to reach an opinion but it is seldom employed to secure drainage.

CONSERVATISM IN THORACENTESIS.

By PAUL H. RINGER, A.B., M.D.,
ASHEVILLE, N. C.

When a patient has a pleural effusion, whether of the so-called primary variety, or whether secondary to a tuberculous process, and when the diagnosis has been made, the paramount question is obviously that of treatment. The time has passed when absorption was attempted by means of purgatives and vesicants, and the only problem that presents itself is: shall thoracentesis be done, and, if so, when?

Pleural effusions can arbitrarily be divided into three classes:

1. Those so minute as to be recognizable with the aid of the roentgenogram or fluoroscope, and not exceeding 250 cc.

2. Those ranging in amount from 250 cc. to 1500 cc., easily recognizable, and, while causing the patient to be conscious of their presence, not giving rise to marked symptoms of respiratory or circulatory embarassment.

3. Those so massive, or else coming on so rapidly as to seriously embarass respiration and circulation.

In the first class, thoracentesis is not considered. In the third class, thoracentesis is imperative. We are therefore mainly concerned with the management of the second class, that of moderate effusions, where discriminating judgment must be exercised.

With the presence of fluid is bound to come the temptation to remove it. We have all tapped chests and obtained from 250 to 1000 cc. of serous fluid, and we have felt pretty well satisfied with our half-hour's work. But I am sure most of us have wondered as time went by, whether we had actually done very much to aid our patient by the removal of the fluid. In fact, why should thoracentesis be done in cases of moderate pleural effusion save for the relief of symptoms and the giving of comfort to the patient?

Paterson, in his quaint, but in many instances practical book on the "Shibboleths of Tuberculosis," states as one of his shibboleths

"That acute serous tuberculous pleural effusions should be aspirated," and in his discussion of this thesis, exhibits far more conservatism than I can feel to be justified, for he refrains from aspirating even the massive effusions unless it becomes necessary so to do in order to save life. Königer, has made a careful study of the influence of pleuritic effusion upon an underlying tuberculous infection. He finds the inflammatory reaction has a decidedly favorable effect on the course of the disease, and often leads to a spontaneous cure. The virtue of the pleurisy lies both in the compression and enforced rest of the lung, and in the production of chemical antibodies which tend to bring about immunity. Upon these grounds he advocates a late tapping, preferably two weeks after the onset in a primary pleurisy. Moderate effusions in a secondary tuberculous pleurisy will often do more good than harm, and does not always need drainage. In an outspoken pulmonary tuberculosis, with cavity formation, the effusion may be allowed to remain indefinitely, or, at least, only a small portion need be removed.

On the other side of the question we find Charles P. Emerson, who, in Tice's Practice of Medicine says the following: "it may be stated that in pleurisy with effusion the fluid should generally be removed as early and as completely as possible and that the tapping should be repeated as frequently as necessary." Emerson, however, goes on, a little later to give Lord's classification of the indications for thoracentesis, which are more conservative. They are as follows: "an effusion large enough to cause definite dislocation of the heart, even though there is no dyspnea; a fluid, moderate in amount, which in two or three weeks presents no change in volume; the presence of cyanosia, of dyspnea or of cardiac weakness, and, lastly, bilateral effusion."

In favor of conservatism in aspirating the fluid, one may advance the arguments that the fluid in itself does no harm, and despite all aseptic precautions, with each aspiration there is danger of introducing infection. The removal of the fluid in no wise affects the pathological process in the pleural membrane whence originates the exudate. It is well-known how frequently an effusion is part and parcel of an underlying parenchymal pulmonary process, for time and again upon getting rid of an effusion a broncho-pneumonia or

pulmonary infiltration has been disclosed. In such cases, the mere mechanical presence of the fluid is beneficial. Effusions arising from other membranes are not dealt with as radically as are those in the pleural cavity. If we happen to diagnose a pericardial effusion, unless it be of large size, we do not at once insert a trocar. A small or moderate amount of fluid in the peritoneal cavity is not at once removed. A knee-joint with fluid in it is held as sacred as was the tomb of Tut-ankh-Amen up to a few months ago. It is the pleural cavity that seems to be the particular stamping ground of tappers.

Now the point is not to find out how many times we have aspirated nor how many cubic centimeters of fluid we have drawn off, but how much good we have done our patients by this procedure. I contend that thoracentesis merely for the sake of taking fluid out of the pleural cavity because some happens to be it, can hardly be justified for the reasons above stated. A consideration of a moderate number of cases should demonstrate whether thoracentesis is of real value as a routine measure. I have accordingly looked up the records of 34 cases of proven pleural effusion occurring in our work. Were I to include the incidence of fluid in cases of artificial pneumathorax, this number would, of course be tremendously increased; but I have not believed it fair to do this, and have taken cases, definitely or suspectedly tuberculous, in whom fluid was present either before or during their stay in Asheville.

Of these 34 cases, 25 were aspirated and 9 were not aspirated. Of the 25 aspirated, 17 did well and 8 did badly. Of the 9 not aspirated, 8 did well and one did badly. Twelve of the 25 that were aspirated had been tapped prior to coming to us, amounts of fluid varying from 150 to 2000 cc having been removed. In 6 of these thoracentesis was done for relief of urgent symtoms. All of these did well. Four of the cases that we tapped were suffering from respiratory and circulatory embarassment. Of these four cases, three did well and one did badly, though a fatal termination did not come about for several months, and then only after many severe hemoptyses. The outcome was wholly independent of the withdrawal of the fluid.

Now what do these few figures show? They show, to all intents and purposes, nothing particularly conclusive one way or the other.

And that thoracentesis does good in the massive cases. They certainly do not show that thoracentesis is indicated as a routine measure any more than they show that thoracentesis increases the gravity of the situation. They do show that it does not make much difference in the moderate effusion whether tapping is done or not. If there is no definite indication that the patient will be benefitted by thoracentesis, there exists no reason for removing the fluid simply because it happens to be present. A massive effusion will always have to be removed, at least in part. A very moderate effusion particularly on the left side, may demand removal. The standard to go by is not the presence of fluid in the abstract, but the general condition and comfort of the patient, and the more one sees of small or moderate effusions the less will be his temptation to rush for the aspirator. The common teaching is that not more than 1000 or 1500 cc should be removed at one sitting. I have no intention of discrediting this elastic and time-honored rule, but I do feel that in those cases of massive or very rapidly appearing effusion, where thoracentesis is undertaken to relieve dyspnea and circulatory failure, no more fluid should be removed than is necessary to attain the desired objctive. James, in his book on Pleurisy, stresses this, saying: "inasmuch as the aspiration is merely for the purpose of relieving the breathing, I do not endeavor to remove any more fluid than is necessary at the time for this purpose." There is no reason to remove all possible fluid simply because the patient does not complain and the aspirating apparatus is working beautifully. It is not a good plan to cause more mediastinal shifting and more change in intrathoracic pressure than is necessary to attain the goal which is the comfort of the patient. Furthermore, it is of course a well-known fact that removal of a relatively small portion of an effusion will not infrequently initiate absorption of the remainder. In dealing with fluid in the chest, as in so many other conditions encountered in the practice of Medicine, I feel that it is far wiser to lead Nature rather than to attempt to drive her.

I have tried the substitution of air for aspirated fluid in too small a number of cases of pleurisy with effusion to have definite views as to the advisability of this procedure. On paper the advantages are numerous and many of the arguments advanced are

aparently sound. In the management of fluid forming during the course of an artificial pneumothorax I am convinced of the desirability of its removal and of the substitution of air, for in these cases maintenance of pulmonary compression and of the integrity of the pleural cavity is essential, but I am by no means sure that this is imperative in simple effusions. In three cases in which substitution of air for fluid was done, two reacted very well and made excellent recoveries while the third went steadily downhill.

The question of thoracentesis, whether to do it or not, if so, when; if not, why not; the amount to remove, the substitution of air, the relative advantages of one large aspiration or of several small ones; all these are moot points upon which the last word has not been said by any means. It is obvious from the title and body of this paper that I advocate conservatism in the use of this most valuable medical operation. There may be, and I rather hope there will be some who will differ radically from the stand I have taken. If so, I can but hope that discussion will serve to clarify the situation as to the management of cases which are often puzzling in the extreme.

DISCUSSION.

Dr. Hawes: Twenty years ago Dr. Shattuck of Boston used to emphasize these facts that Dr. Ringer has brought out, and urged his students not to try to drain the last drop of fluid out of the chest.

Dr. Pryor: It should be emphasized that aspiration of the pleura is always more or less dangerous and I have tried to emphasize this fact repeatedly. On five different occasions patients have collapsed in a most dangerous manner upon the introduction of the needle. Some of the unsatisfactory results, as the result of aspiration are not so much in my opinion due to the amount of fluid withdrawn as the rapidity with which it is removed. The fluid should be aspirated slowly. I have seen one case of a severe broncho-orœa following rapid withdrawal of fluid. I think the requirements are the slow draining off of the fluid, not attempting to remove it all and considering each individual case. It is impossible to draw off all the fluid; some is always left in the costal sinuses. This has been demonstrated at Perrysburg by fluoroscoping immediately after an attempt to entirely empty the pleura.

Experiments on rabbits have confirmed the observation that the pleural cavity, even if desirable, cannot be entirely drained and there is always some re-accumulation of the fluid within twelve hours after attempts at complete removal.

DR. HALL: Tapping the chest is dangerous. One thing that is more dangerous is not to tap if there is fluid. When we draw off one effusion, if the patient is not well, it is time to try again and see what it is.

DR. DUNN: I would like to call attention to one class of patients. I refer particularly to those in whom full growth has not been attained. These cases with effusion should be watched very closely where there is delayed absorption, fluid not being allowed to remain to the formation of large organized exudates, which, with the subsequent cicatricial contraction, bring about fixation and sometimes deformity of the chest.

DR. ELLIOTT: What is the basis on which we decide whether to take a large amount or small amount? If we were a little more careful to think of pleural pressures we might decide when to stop. I think the aspirators are a source of danger. With the aspirator the man has no idea what suction pressure is used, and I think it would be safe to discard the aspirator and use simple siphonage. I think when we get to the point that the fluid will not run from the chest, when the end of the tubing is two inches or one inch below the opening in the chest, it is time to stop.

DR. LLOYD: I agree with pratically everything Dr. Ringer said. I think each case should be handled as that paricular case indicates. I prefer to aspirate a larger quantity at longer intervals. Sticking a needle into the chest is not a simple procedure and I feel that we should do it as infrequently at possible.

DR. RINGER: I wish to thank Dr. Elliott for bringing up the question of the amount of suction to be exerted during aspiration. It is a great temptation to have the huskiest person around operating the pump of the aspirating apparatus and keep urging them to make a good vacuum so that the fluid runs in a rapid, steady stream. This is a bad plan, as the suction power exerted is tremendous and the effect on the patient can often be unpleasant. I never try to have more vacuum than is necessary to make the fluid run steadily and slowly. There is no objection to having the fluid come out of the chest slowly. In fact it is far better than to have it squirt out.

Dr. Hall's remarks in regard to needling are really not in line with the discussion of this paper, as the title is "Conservatism in Thoracentesis" and not a discussion of the question of exploratory puncture. While of course no one should desist from exploratory puncture when there is any good and sufficient reason to do it, I feel that I must deplore the tendency to stick needles in here, there, and everywhere. It is always a certain shock to an individual to have a needle stuck into the pleural membrane and I think that with careful physical examination and careful Roentgenological investigation, the number of exploratory punctures should be markedly reduced to the benefit of the doctor in enabling him to make diagnoses without resort to this mechanical method, and to the benefit of the patient in keeping him from having to be subjected to an unpleasant procedure.

TREATMENT OF LARYNGEAL TUBERCULOSIS IN SANATORIA

By C. D. PARFITT, M.D.,
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A year ago, through a questionnaire sent to twenty sanatoria in Canada, I made an inquiry regarding the amount of laryngeal tuberculosis recognized and the routine of examination and treatment employed. Of 8393 cases, 5.4% actually had tuberculous laryngitis. Since the numbers of patients in the different institutions varied greatly, the average of the percentages of each institution probably gives a truer estimate—this was 8.6%. In thirteen sanatoria discrimination against the admission of cases with tuberculous laryngitis was probably exercised; these averaged only 4.5%, while in six institutions in which there was no selection, the average was 16.6%. It was striking that in two adjoining provinces with large provincial institutions, in one the average was 33% and in the other 3.2%.

Besides discrimination against the cases with laryngeal tuberculosis, no doubt the general class of patient admitted varied, as did the criteria of diagnosis and the thoroughness of routine examination. Because of these last two variables, 8.6% is probably too low. In four institutions in Massachusetts, Hawes[1] found the incidence of laryngeal tuberculosis to be 8%. At Mount Vernon Sanatorium Barwell[2] found 11%; at the King Edward Sanatorium Sir St. Clair Thomson, 22%[3]; and at Otisville Sanatorium Dworetzky 25.6%[4]. At the large tuberculosis clinic of the Gage Institute[5], Toronto, only 1.2% of the patients examined by larynologists had laryngeal tuberculosis, while at the Phipps Institute in Philadelphia 7.4% showed laryngeal tuberculosis. Here probably admission to the hospital also went through the clinic.

In twenty Canadian Sanatoria examination of the larynx on admission and as routine was undertaken in 8; on admission and suspicion in 6; on admission only in 2; and on suspicion only in 4 A con-

sulting laryngologist visited at regular intervals of from one week to two months, in 4, and irregular intervals in 6. In 10 there was no consulting laryngologist on the staff. Adequate facilities for treatment were found in 1; incomplete facilities in 4; and 9 depended upon a local specialist.

In regard to treatment, silence, sprays and insufflations were used in all; intratracheal injections in 6; pigments in 11; chaulmoogra oil in 2; natural heliotherapy in 12, artificial in 2; tuberculin in 5; nerve blocking in 7; the galvano-cautery in 2; curettage in 1; epiglottidectomy in 4; other operations in 3.

In 2857 cases of phthisis, recorded by four authors, quoted by Lockard[7], the average of laryngeal involvement was 29%. Horne[8] in 359 consecutive cases of phthisis found no change in only 9, while 55 he considered would escape the naked eye at post-mortem, i. e., 97.5% were tuberculous. Fetterolf[9] found 83% of cases coming to autopsy at the Phipps Institute had the larynx involved. At Brompton Hospital[10] 52.6% showed naked eye evidence of tuberculosis at post-mortem. By miscroscopical section even higher percentages than these have been shown to be tuberculous.

St. Clair Thomson's[11] comparison of the mortality in sanatorium patients without and with laryngeal tuberculosis from three to seven years after discharge, emphasizes the importance of this complication in prognosis. In contrasted groups laryngeal tuberculosis makes the prognosis relatively more gloomy for the earlier pulmonary cases than the moderately advanced, and more so for these than for the far advanced. Without this complication the expectation in fairly early cases is that 60% of patients will be alive in from three to seven years after discharge from the sanatorium. In similar groups of pulmonary cases the complication reduces those living to 30%. For prognosis a laryngeal examination may outweigh in importance all other clinical examinations. While not necessarily the fatal disease of a generation ago he considers laryngeal tuberculosis next to meningitis the most serious complication of pulmonary tuberculosis.

Within recent years Doctors Minor[12], Casselberry [13] and Forster[14] have given papers before this society on this subject, urging both early diagnosis and efficient methods of treatment. From the literature one is forced to conclude that outside the larger health

resorts the treatment of laryngeal tuberculosis has not been energetic. Yet the literature shows that much may be done for such cases. Specialists are perhaps quite properly critical of amateurs entering their field. Those sanatoria, however, which are isolated and which cannot have the convenience of consultations and regular visits from specialists must perforce undertake the treatment of laryngeal tuberculosis by members of staff, even operative procedures, if these cases are to get their due. With reasonable restriction in regard to the hopeless, this group of cases should be admitted to sanatoria where they can best be treated, since frequent visits to an office may be prejudicial to their recovery.

During the period 1909-1922, of 652 tuberculosis patients treated in a sanatorium 107 (16.4%) had laryngeal tuberculosis. Laryngitis considered to be non-tuberculous occured in 9.5%. The following tables briefly summarize points of interest in a complex annalysis:

Patients tuberculous	652	100%
Non-tuberculous laryngitis	59	9.5%
Laryngeal tuberculosis	107	16.4%

CLASSIFICATION N. T. A.

	Whole Series.	Tb. Lx. Group.	
Type—General	%	%	
Incipient	28	0	(T.I — 2)
Moderately advanced	22	24	(T.II —22)
Far advanced	50	76	(T.III—76)
Result—General			
Arrested	5	0	
Apparently arrested	14	3	
Quiescent	20	8	
Improved	41	44	
Unimproved	17	38	
Died	3	7	
Result of Treatment Lx. Lesion			
	%	%	
Apparently cured	6		
Arrested	1	26	
Apparently arrested	19		
Quiescent	7		
Improved	20	27	
Stationary	25	25	
Unimproved	17		
Died	5	22	

Classification of Major Lx. Lesion

Type	%	Character. Congestion	Infiltration	Ulceration	Hypertrophy
Mild . . .	56	7	17	17	15
Moderate .	25	..	7	18	..
Severe . .	15	..	..	14	1
Healed . .	4	..	..	..	..
		7	24	49	16

Analysis of Lesions

362 lesions (358 intra laryngeal) recorded in 114 cases (7 twice included).

Site	% Total	Cong.	Infilt.	Ulc.	Hyper.	Paresis.	Fixation
Epiglottis . .	6.7	1.7	2.5	2.5	..	..	..
Ary.-Epiglottic Folds . . .	2.0	.3	1.7	..	..	..	..
Ventricular Bands	12.5	3.4	5.8	3.0	.3	..	..
Vocal Cords .	34.4	13.6	7.0	10.0	1.9	2.8	..
Arytenoids . .	11.3	3.1	3.9	3.4	.8		.3
Inter-Ary. Space	25.9	3.4	10.8	5.8	5.8	..	..
Ant. Commissure	.3	..	..	.3	..	..	..
Subglottic . .	5.6	.5	4.5	.5	..	..	..
% Total . .	100	25.9	36.2	25.9	8.9	2.8	.3

The pharynx was involved in one case (.3%) and glands in three cases (.9%).

Methods of Treatment (exclusive of voice rest and sprays, used in the great majority):

Intratracheal injections in 1 (+), pigments in 21 (+), reflected sunlight in 3, galvano-cautery in 17, operations in 9 (curetting 4, arytenoidectomy 1, epiglottidectomy 4, trachœtomy 1), and nerve blocking in 4 cases. As an auxiliary tuberculin was used in 18, and artificial pneumothorax in 14 cases.

For more than twenty years I have been so placed that regular visits by specialists were quite impracticable, and I have been obliged to attempt the treatment of these cases myself. Records for the first seven years are lost to me. During this period pigments, especially formalin, with occasional curettements, were consistently used, with fair results, effecting cures in a number of cases and restraining progress of the lesion in many others. Except in very mild cases I have never felt it right to rely upon sanatorium treatment alone when it seemed possible to accomplish something by local treatment.

The daily application of pigments is very exacting in the matter of time. This is well illustrated in Bulloch's[15] large series, which, however, show what remarkable results can be obtained by such persistent treatment. During the last ten years, however, I have used the galvano-cautery consistently where applicable, and this, with silence, has made it possible to dispense almost entirely with

other time-consuming measures. It has, as Wood[16] expresses it, "largely displaced bloody surgery," and the results are frequently quite dramatic. It is a practicable and relatively easy method of treatment, although its adoption has been slow. It was, I believe, first used for laryngeal tuberculosis by Voltolini[17] in 1861 and again by Heryng about 1887. Dundas Grant[18] advocated it at the beginning of this century, but was little used until Mermod's[19] and Gruenwald's[20] publications in 1903 and 1907 respectively. As early as 1904 Harland[21] reported its use at the Phipps Institute. Thomson[22] found that while sanatorium treatment with silence arrested the local lesion in 12.5% of his whole series, the galvano-cautery added 8.5% to the arrested group. He applied it to 20% of the whole series and secured arrest in 42% of the cases thus treated. Its adoption should be general in all sanatoria. Of 17 cases of my series its use was only intended to be palliative in 3; these patients died. In 2 the condition remained stationery; in 2 there was material improvement; and in 10 (59% of the cases thus treated) the result was either apparent cure or arrest of the lesion. It has been used in a number of other cases not included in this series. The appended case histories will illustrate its value as the major factor in promoting recovery from the laryngeal complications.

In the sanatorium the patient with laryngeal tuberculosis has living conditions and a regime with which local measures give much more promise of benefit than when they are carried out without these advantages. Atmospheric conditions are improved and the irritation of tobacco and alcohol controlled. Rest of voice, even silence, can be carried out with daily encouragement and with the maintenance of some companionship. The conditions above the larynx which further laryngeal irritation emphasized by all writers, can be investigated, referred if necessary to a rhinologist and treated as a matter of routine. Such treatment may lesson irritation and cough surprisingly. Cough may, even more than talking, aggravate the lesion. Its cause should be carefully sought. Liquifying expectorants and sedatives are usually necessary for a time. Cleansing and sedative Sprays, sedative intratracheal injections and insufflations can be undertaken at frequent intervals as a routine and given effectively. Instruction and supervision in the use of reflected sunlight are easily

managed. This method of treatment has greatly reduced aprehension in regard to definite or suspected laryngeal tuberculosis in our western provinces, where it can be promptly and efficiently applied.

More active measures will be frequently indicated. While procrastination in their application will sometimes be desirable, prompt active interference may gain greater security for the patient and save him much time and apprehension, and also save both the physician and staff much time. The swabbing with pigments while condemned by some, has given marked success to others who have patiently followed this method, I am convinced of their value but now seldom use them. Frequent applications of formalin and lactic acid have been displaced by the occasional use of Lake's[23] pigment (lactic acid 50, formalin 7, carbolic acid 10, water 33, or by the galvanocautery. Such applications are best made with a curved applicator of tapered brass wire, which is flexible, yet springy, and is less likely to do injury than are the rigid applicators usually listed by the instrument makers. After a moderate amount of practice on an improvised dummy the consistent application of formalin (3-10%), lactic acid, or iodoglycerine solution will in localized ulcerations have some beneficial effect, at least in lessening secondary infections, and at the same time be excellent training for endo-laryngeal operations. Then the occasional application of Henryng's curettes, while useful in itself by removing infected tissue, will help to make applications of pigments more effective.

These curettes are also of use in the removal of granulomata and tuberculomata. Here the double curettes or punch forceps of Lake or Watson Williams are more effective. The epiglottis punch-forceps of Lake or Barwell, with or without the fixation forceps of Hett, can remove the epiglottis with one punch and give the greatest relief to odynophagia when due to a diseased epilogottis. Escat's tongue retractor is a help for this relatively simple operation. After such removal pain may remain because of a swollen arytenoid, which may also be relieved by the removal of a portion of it with Lake's forceps or preferably by ignipuncture. The latter method is also urged by Wood as superior to and safer than epiglottidectomy. Indeed the galvanocautery has fairly displaced all operative meas-

ures, possibly excepting that of epiglottidectomy and that for the preliminary removal of papillomata with bases of limited extent.

The electric cautery is not difficult to use when reasonable dexterity in making topical applications has been acquired. The point can be placed while cold but must be removed while hot. On removal there is the easy possibility of scorching the laryngeal surface with the heel of the cautery. Dan McKenzie has designed a set of five points set at different angles, which I have been accustomed to use, with a most convenient handle designed by Hovell. Wood describes three types of points with which I am unfamiliar.

For endo-laryngeal operations an hour is chosen as far as possible from a meal. An injection of morphia gr. 1/6 and atropin gr. 1/150 is given twenty minutes before anaesthetization is begun. The Pharynx is lightly sprayed with 1% cocaine solution. Ten drops of 10% cocaine solution is measured into a small conical glass to which a drop or two of adrenalin solution is added. Two drops are dropped upon the papillae at the base of the tongue, also upon the epiglottis, and into the larynx while the patient phonates. The remainder, two drops at a time, is placed upon the site of cauterization at intervals of two minutes. A Hartwell's droper is most convenient. The patient is required to expectorate the solution promptly. A part of a measured grain of cocaine crystals is then applied on a moistened swab directly to the site and held there; this is repeated in three minutes and after another three minutes cauterization or operation is begun. Experiment may show that less cocainization will suffice. The preliminary hypodermic greatly lessens mucus besides being sedative to the patient in every way. After the operation oil sprays or intratracheal injections of sedative oil solution and the throat ice-bag give comfort.

The object of this method of treatment is not merely the destruction of a certain amount of tuberculous tissue but the production of a reaction, resulting in improved blood supply and the formation of scar tissue. The disappearance of tubercle in the cauterized and surrounding tissues has been shown experimentally. The patient must have enough vitality to react sufficiently to repair the injury done by the cautery. With such reactive power there is no contra-indication to cauterization as regards the pulmonary lesion and gen-

eral condition. The first cauterization should be cautiously undertaken and limited when the general condition is much impaired. At all times restraint should be exercised in regard to the number of punctures and amount of surface attacked. The depth of puncture must be controlled in order to avoid burning cartilage. It is best to do less than one may feel inclined to do. Experience only can teach the extent and depth of cauterization for different types of lesions and patients. The same area should not be again attacked until healing has taken place and opportunity for shrinkage given. This will require about a month. Other areas may meanwhile be treated. The reaction is relatively slight. Only once have I seen distress from dyspnoea, when cauterization was combined with a cutting operation in a small larynx. The patient rarely suffers at the time and is not fearful of a repetition of the treatment. Soreness afterwards subsides quickly and is rarely severe. Wood[16] believes that all types of localized tuberculosis lesion within reach will be more successfully combatted by cauterization than by any other form of treatment and that 90% of incipient cases can be cured.

Active treatment is to be preferred, as a rule, to delay when the lesion is early even though the lung disease is probably curable. Active treatment is necessary in grave cases for the relief of pain and sometimes for dyspnoea, then extreme weakness is the only contraindication. When there are no severe symptoms in acute and far advanced cases operative treatment is unnecessary and probably prejudicial. As a rule the higher the disease is situated in the larynx the more urgent is the need for energetic treatment, because of the likelihood of rapid extensive infiltration.

For palliative treatment, Leduc's autoinsufflator for anaesthetic and antiseptic powders, Yankauer's dropper for solutions, and Seymour Jones' U-auto-injector for intratracheal injections of solutions in oil are most useful. The last mentioned is used by inserting a catheter through a nostril, the point of which behind the uvula immediately overhangs the larynx. Nerve blocking has given relief, sometimes very marked, in about 60% of the attempts. It has been repeated a number of times on the same patient. Epiglottidectomy may also be necessary even with successful nerve blocking, to give

full relief. Feeding, and posture when taking food, demand much consideration.

In the sanatorium, laryngeal tuberculosis has not proved fatal in cases which otherwise had a favorable prognosis. It has been the least dangerous complication of pulmonary tuberculosis and the one most amendable to treatment. It has often become arrested or cured before the patient had recovered from the pulmonary disease. Even in cases entering with advanced laryngeal lesions and bad prognosis, relative comfort can frequently be given. To be able to change certain disaster to relative or complete recovery by a little skilful interference is such a satisfaction that the treatment of laryngeal tuberculosis in sanatoria deserves every consideration.

CASE HISTORIES

M. 51. Mr. G. McG. Age 26. Admitted 17/5/15. Discharged 26/8/15. Duration of illness—two years. Early symptoms—hoarseness no pain. Nine months prior to admission hoarseness marked. Galvano-cauterization by larynologist; later swabbing. Six months prior to admission entered a sanatorium; rest and sprays. In two months time transferred to hospital for advanced cases, where he received no local treatment.

Far advanced; Turban 3, R3, L2. B. t. positive; comparatively afebrile.

Larynx—Patient can only whisper. Both ventricular bands infiltrated and granulomatous; on the right a small ulcer, the right cord is ulcerated in the middle of the superior surface and inner margin; the left cord is infiltrated and granular. Two months later a small papilloma developed near the right vocal angle in the interarytenoid space.

Galvano-cautery was applied three times in two months. Seven months after the last application the larynx was clear of ulceration and thickening of the cords much reduced; very slight thickening of the i. a. s. Two years after cessation of treatment the cords were slightly thickened, no irregularity, slightly congested. The voice, while often husky, was sometimes quite clear. The general condition was good and the lungs themselves had made general improvement.

M. 58.—Mr. H. P. B. Age 31. Admitted 17/5/15. Discharged 2/4/17. Pleurisy with effusion 1903. Recent breakdown December 1914. January 1915, throat sore, odynophagia, huskiness; recent hemoptysis; constitutional symtoms marked. Far advanced; Turban 3, R2, L3.

Larynx—Right cord thickened, large granuloma at middle. Infiltration of left arytenoid. Slight ulceration in interarytenoid space. 1/6/15 Galvano cauterization right cord and left arytenoid. 24/6/15 Both ventricular bands swollen. 3/7/15 Cauterization of left ventricular band and left arytenoid. 15/8/15 Cauterization of left arytenoid; application of Lake's solution to ulcer which was followed by severe reaction. 15/9/14 Throat improved; relative fixation of left cord. 14/12/15 Superficial ulceration of the right cord and moderate congestion. Left ventricular band thickened probably ulcerated; left cord irregularly thickened and congested. Less soreness in larynx. 18/3/16 Cauterization of left ventricular band and recently infiltrated right arytenoid. 22/3/16 Cauterization of interarytenoid space. 18/5/16 Cauterization of interarytenoid space. 24/10/16 A small papilla in the centre of the i. a. s. Slight fissure in the right ventricular band; the left cord fixed, not congested, overshadowed by the left ventricular band; scar on the left arytenoid. 24/2/17 Mucous membrane of very normal appearance. Left cord fixed, but few sensations referred to the larynx. Patient has used voice for three months—fair quality when not strained. Marked general and pulmonary improvement.—1923. The patient has been carrying on his occupation as salesman since discharge; uses voice a great deal; no recurrence of laryngeal lesion.

M. 67. Mr. G. N. Age 17. Admitted 1/2/16. Discharged 29/9/16. Duration of disease 3½ years. Ischio-rectal abscess. Hoarseness four months prior to admission. Far advanced; Turban 3; R1, L2; moderately febrile.

Larynx—Ulceration left p. vocalis. 22/3/16 Left cord irregularly ulcerated and granular. Left ventricular band ulcerated, and ulcer at base of epiglottis, left side. Much general congestion. Galvano-cauterization. 24/4/16 Immediate and great improvement following cauterization. Cauterization repeated. 8/6/16 Artificial pneumothorax induced on left side. 4/12/16 No ulceration in

larynx; some thickening and congestion especially on the left side. Small papilloma in i. a. s.

Three years after second cauterization nothing noteworthy in the throat. Patient was very well. Comparatively recently he was very well and at work.

C. 36.—Mrs. B. T. P. Age 38. Admitted 24/2/18. Discharged 4/6/20. Duration of disease fourteen years. Variable laryngeal symptoms during this time. Present illness began eight months before admission. Symptoms general; also hoarseness, almost complete aphonia. Far advanced; B. Turban 3; R1, L2, with cavity.

Larynx—Hoarseness, Lancinating pains in right ear and odynophagia; moderate papilloma; ulcerated at apex, on left side of i. a. s. 24/3/18 Papilloma cauterized. 30/4/18 Cauterized 21/5/15 Healthy granulations; throat has not been so comfortable in years. Six months after first application only small elevated scar was to be seen. When seen three years later, no recurrence of trouble; despite fact that there had been a gradual enlargement of cavity in left lung, attempted pneumothorax had been unsuccessful. While patient has made great improvement in weight, symptoms of pulmonary activity are induced by the slightest liberty of movement; she continues to remain on most complete rest.

C. 20.—Mrs. J. B. Age 53. Admitted 14/12/17. Discharged 3/4/18. Duration of present illness five months; began with cold and hoarsness. Moderately advanced; C. Turban 2; R1, L2. B. t. positive.

Larynx—Marked huskiness; both cords eroded along free margin, involving the vocal process. Ulceration specially marked over the superior surface of the left cord. Surfaces of these ulcers cauterized lightly four times, during ten weeks. When patient left there were no ulcers to be seen; moderate congestion of both cords remained. Very good voice could be produced. Four years later patient relapsed—died five years after discharge. No recurrence of throat condition.

DISCUSSION.

Dr. Miller: Does Dr. Parfitt use his methods in acute cases or chronic cases?

PRESIDENT: He has given the methods of treatment in these cases. Sanatorium treatment of cases is decidedly the more practicable. The treatment of individuals out of a sanatorium is largely unsatisfactory. In that work, with practically ambulatory patients, many laryngologists prefer the clean cut wound, rather than by the galvanocautery, because the work is cleaner and it leaves a surface which heals more naturally than that produced by a cautery. A cautery is not only a wound but a burn, so that is the reason why most laryngologists prefer the clean cutting instruments than the galvanocautery. I am delighted to hear Dr. Parfitt's paper.

DR. PARFITT: In reply to Dr. Miller, I have found the cautery may be successfully used to make the patient more comfortable. One must be cautious how much injury he creates. In cases that have a reasonable degree of resistance, I think the galvanocautery is very useful, except in those cases with pulmonary involvement and whose progress is down-hill.

FREQUENCY OF TUBERCULOSIS INFECTION OF THE LUNGS ASSOCIATED WITH SO-CALLED SURGICAL TUBERCULOSIS AND THE BEHAVIOR OF THE PULMONARY LESIONS.

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It has long been the custom in the medical profession to diagnose and treat tuberculous lesions of an organ or structure other than that of the lungs as a localized disease where an operative procedure has been too frequently considered the only treatment, giving scant thought to the fact that the lesion is often only a local manifestation of a manifest or obscure tuberculosis in the lungs or lymphnodes. Because of this accepted view implying as it does a failure to acknowledge the almost universal co-existence of a thoracic lesion in this type of tuberculosis, the term surgical is applied. The term being descriptive, its application focuses attention upon local manifestations and an attack upon them alone.

The usual mode of infection and sequence of events following infection being known it seems inconsistent to apply a term which has become misleading and suggestive of a form of treatment which is rapidly being discarded. The term surgical tuberculosis has become a misnomer and should be abandoned as largely obsolete. Treatment should be confined to conservative and constructive and not destructive measures. The terms non-pulmonary or extra-pulmonary tuberculosis are open to criticism and lead to a false conception of the true pathology of tuberculosis. We must either find a new descriptive term to differentiate between pulmonary tuberculosis and other forms or include them all for hygenic reasons in one group and thus recognize the pathological and clinical unity of tuberculosis lesions, and at the same time accept a constitutional idea of treatment.

So-called surgical tuberculous lesions, with few exceptions are due to secondary invasions. We are learning to regard tuberculosis as a disease characterized by many local involvements no matter whether the lesion apears in joint, bone gland, kidney or lung. A primary outbreak of clinical tuberculosis most often emerges from an old focus in the glandular tissue or the lung, and it often follows a tuberculous implantation of early childhood.

Statistics at the J. N. Adam Memorial Hospital, confined to bone and joint tuberculosis reveal that 50% of lesions in adults and 44% of lesions in children exhibited sinuses and secondary infection on admission, and also that 36% of the bone and joint lesions in adults and 27% in children had undergone operations before admission.

A thorough history and a careful examination in most instances will reveal the previous or present existence of a pulmonary lesion. The recognition of the secondary nature of localized outbreaks in tuberculosis will most frequently lead to the detection of the primary focus and will indicate the necessity of a constitutional hygenic treatment. It is becoming more and more apparent that the patient must be treated as a whole in observance of measures which promote general improvement and increase resistance in all forms of tuberculosis. Histories of many of the so-called surgical cases at the J. N. Adam Memorial Hospital disclose the fact that the examination of the chest either has been a very cursory affair or has been entirely neglected. Disease of the lung was not searched for and the diagnosis of tuberculosis was based on the obvious local lesion. Not a few have been found to be in a moderately advanced stage of the disease with positive sputum. This is mentioned to emphasize how deep rooted is the prevailing opinion that tuberculous lesions are disease entities and not manifestations of multiple invasion.

The literature on orthopedic surgery now displays abundant testimony in favor of conservative and constructive treatment. Heliotherapy has been accorded the strongest advocacy because of the recognition that has been freely given to the conception that localized tuberculous lesions are often only evidence of a disease located elsewhere, and that the primary focus should be looked for in the lungs or glands. Surgeons accepting this conception of the disease

and disappointed in the results of surgical interference do not operate with the same enthusiasm in this type of tuberculosis as they did before the wonderful results obtained by Heliotherapy had been brought to their attention. Dr. Sorrel, Chief Surgeon of the Municipal Hospital of Paris, has arrived at the conclusion that Heliotherapy is the most valuable treatment for bone, joint and glandular tuberculosis. His opinion is typical of many progressive surgeons who have largely discontinued radical procedures which were formerly unwisely employed.

Paranthetically, it seems fair to assume that in the near future the accepted treatment for localized lesions will consist of the four dominating factors.

1. Heliotherapy.
2. Rest.
3. Full Nutrition.
4. Open air life with air bath and instead of fixation or operation to secure anchylosis such orthopedic rest and exercise necessary to obtain motion and restore function.

Very little definite information can be gathered from the literature as to the occurence of clinical, pulmonary tuberculosis associated with the disease in other parts of the body. This may be accounted for by the following factors:—

1. Because attention is riveted upon the local lesion.
2. Failure to search for pulmonary lesions.
3. Failure to recognize thoracic disease due to lack of expert knowledge.
4. Failure to correlate the findings.

Some available statistics are compiled from post-mortem findings and these give very little information except the general one that cases which succumb to surgical tuberculosis almost invariably show evidence of a tuberculous lesion in other parts of the body, and that the greatest number exhibit pulmonary involvement. It is also shown that the thoracic foci supply evidence of being the oldest established lesions and the site of the primary invasion.

Osler reports in 275 autopsies where a surgical lesion was present, a pulmonary involvement in 99%. E. Konig found on post-

mortem, that 79% of the fatal cases of bone and joint tuberculosis had old tuberculosis foci in the lungs or glands.

Van Zwaluwenburg, Wickett and Merrill in a series of 111 cases of extra-pulmonary tuberculosis found x-ray evidence of pulmonary complications in 62%. Unquestionably, this proportion is too low as the careful expert pathologist can detect many lesions which would otherwise escape observation.

Statistics of the J. N. Adam Memorial Hospital show that 967 cases of surgical tuberculosis have been discharged. Of this number 420 were adults and 547 children. Seventy-one percent of the adult group and 18% of the children showed a manifest tuberculosis in the lungs as evidenced by physical signs, x-ray and laboratory reports. Undoubtedly the percentage of positive results in the children is apparently low when we consider how difficult it is to detect the pulmonary lesion in the child. In the adults the highest percentage of pulmonary tuberculosis was in association with rectal fistula, viz: 88% and in tuberculosis of the genito-urinary system 80%. The next most frequent evidence of tuberculosis was found with peritoneal tuberculosis—75%; gland tuberculosis exhibited a pulmonary lesion in 68% and in association with bone and joint tuberculosis 68% showed involvement of the lung. Among the children a demonstrable pulmonary lesion was present in 83% of genito urinary tuberculosis. In peritoneal tuberculosis a pulmonary lesion was present in 32%. In the gland cases 18% and in bone and joint cases 14%.

It will be noted that there is a parallel sequence in the incidence of pulmonary involvement in adults and children and that the thoracic disease is most frequently discovered in genito urinary tuberculosis and least frequent in bone and joint disease if the rarer manifestations are not considered.

An interesting point not presented in the statistical table is that 14% of the bone and joint cases in adults and 21% of the same class of cases in children presented multiple tuberculosis lesions. Contrary to expectation, pulmonary tuberculosis was found in 65% of those cases that had multiple lesions and 70% in those with single lesions. It is also noted that a large number of cases that present a high lumbar or low dorsal tuberculosis the kidney was also involved.

The clinical fact is observed that in those cases showing both

a definite pulmonary tuberculosis and an involvement of another organ or structure is that the pulmonary focus very rarely manifests itself as an acute, active or rapidly progressive lesion, but in the great majority of instances presents itself clinically as a mild quiesent non-progressive lesion in which arrest or healing occurs more rapidly than in those cases limited to a pulmonary invasion. Of the whole number of localized lesions associated with pulmonary disease only ten or 2½% of the cases pursued an acute or progressive course. We are satisfied that the clinical observation is correct that a pulmonary lesion is in some way modified in its course by associated lesions in the body. It is our hope that continued study may aid in explaining this observation.

In conclusion, the purpose of this brief contribution is to emphasize the fact that the vast majority of localized tuberculous lesions in any part of the body are associated with evidence of some form of pulmonary tuberculosis disease and that this vital truth should be recognized to encourage the employment of general hygenic measures to secure results far superior to those which have been obtained in the past. The plea is for the use of rational, conservative and natural measures based upon the advanced knowledge of pathological and clinical facts.

DISCUSSION

DR. EDSON: The differentiation between tuberculosis in the lungs and tuberculosis in the hylus, tuberculosis in the glands, and tuberculosis in the bronchial tissues, is an interesting study pathologically and anatomically. Before we had x-ray pictures patients were constantly running to us and we said it was bronchitis, not tuberculosis, and that it was their bronchial tubes affected. We know the dangers that come to these patients, because it was only bronchitis. Now under roentgenogram studies we are hearing more and more we must differentiate between pulmonary tuberculosis and hylus tuberculosis from the point of view of the patient. That is a dangerous discrimination. We may know that hylus infection has a slower progress, a better prognosis. We know that the progress is to his advantage and we do not have to keep him as quiet, but we are going to make a clinical error if we let this new knowledge out among them too soon.

DR. DUNHAM: Dr. Pryor speaks of lung lesions and lesions of the root of the lung. I wish he would tell us how many of those were definitely pulmonary.

DR. PRYOR: In reply to Dr. Edson, I do not belong to the group which is always positive and sometimes accurate. I am sufficiently pessimistic in

MOST COMMONLY ATTACKED ORGANS OR STRUCTURES. THE FREQUENCY OF THEIR INVOLVEMENT AND PERCENTAGE OF PULMONARY COMPLICATIONS IN SO-CALLED SURGICAL TUBERCULOSIS

	Adults		Children		Totals	
Diagnosis	Cases	With Pul. Lesions	Cases	With Pul. Lesions	Cases	With Pul. Lesions
Skin	4 or57.1%	1 or 25%	3 or 42.8%		7 or .79%	1 or 14.2%
Eye	7 or 33.3%	6 or 85.7%	14 or 66.6%		21 or 2.3%	14 or 28.5%
Glands	95 or 21.3%	65 or 68.4%	351 or 78.7%	63 or 17.9%	446 or 50.8%	128 or 28.7%
Bone and Joints	169 or 56.3%	117 or 68.1%	131 or 43.7%	18 or 13.7%	300 or 34.2%	135 or 45%
Peritoneum	69 or 61%	51 or 73.9%	44 or 38.9%	14 or 31.8%	113 or 12.8%	65 or 57.5%
Kidney	35 or 92.1%	27 or 77.1%	3 or 7.9%	2 or 66.6%	38 or 4.3%	29 or 73.6%
Testicle	18 or 100%	14 or 77%			18 or 2%	14 or 77.7%
Bladder	5 or 83.3%	4 or 66.6%	1 or 16.6%	1 or 100%	6 or .68%	5 or 83.8%
Rectal Fistula	17 or 100%	15 or 88%			17 or 1.9%	15 or 88%
Prostate	1 or 100%	1 or 100%			1 or .11%	1 or 100%
Grand Total	420 or 43.4%	301 or 71.5%	547 or 56.4%	98 or 17.9%	967 or 100%	399 or 41.2%

accepting interpretations of x-ray findings of the chest. These cases included a statistical table showed positive signs of tuberculosis.

The reason we do not report more fully regarding tuberculosis of the lungs in children is that we share the belief of many others that diagnosis of tuberculosis in the child by x-ray or physical examination is very difficult and uncertain.

In reply to the question concerning the location of the disease; it was always found in the lung tissue. It is dangerous to state that a cure has resulted from the disappearance of cervical glands by x-ray treatment or surgical procedure. The enlarged glands may disappear but we have repeatedly found revealed in the x-ray plate evidence of large bronchial nodes which persisted.

WHAT ARE THE MOST RELIABLE AUSCULTATORY SIGNS IN THE DIAGNOSIS OF CLINICAL PULMONARY TUBERCULOSIS?

By FRED H. HEISE, M. D.
TRUDEAU SANATORIUM

Physical signs undoubtedly play an important role in the diagnosis of clinical pulmonary tuberculosis. However, the relative values of the various signs heard during auscultation have not been definitely determined upon nor largely accepted by the profession. For this reason I have analyzed a series of cases to see to what extent, when my methods and standards of interpretation were used, such changes in breathing as moderate broncho-vesicular, distant, exaggerated or broncho-vesicular breathing with increased vocal resonance (whispered voice) when occurring without the presence of rales were accompanied by definite parenchymatous changes in the x-ray and a definite diagnosis of clinical pulmonary tuberculosis. In a similar manner the presence of rales, regardless of breath sounds, was analysed.

Perhaps not many clinicians would interpret the physical signs heard upon auscultation in any single case in an indentical way unless the changes were quite marked. Many clinicians likewise determine the auscultatory physical signs under differing methods of breathing. However, most examiners have a standard in their own mind at least a standard of interpretation which is arrived at through a standard method of examination. The individuals in this analysis were examined for breath sounds in the sitting position on a stool and were told to breathe through their mouth. No effort was made to control intensity or rhythm.

All breath sounds were interpreted as normal unless it was thought some pathological pulmonary condition existed. The cases were divided into the following groups:

1. Those in which supposedly abnormal broncho-vesicular

breathing usually accompanied by an increase in whispered voice occurred without rales. This group was composed of 248 cases.

2. Those in which there were no abnormal breath sounds nor rales, 45 cases.

3. Those having distant or exaggerated breathing with no rales, 13 cases.

4. Those in which moderately coarse or fine rales were indefinitely present—questionable rales—the breath sounds being disregarded, 36 cases.

5. Those in which rales were heard but not in the upper one-third—breath sounds disregarded, 93 cases.

6. Those in which fine rales were heard in the upper one-third, with rales present or absent elsewhere—breath sounds disregarded—140 cases.

7. Those in which moderately coarse rales were heard in the upper one-third with rales present or absent elsewhere—breath sounds disregarded—425 cases.

In group 1 (broncho-vesicular breathing) composed of 248 cases, clinical pulmonary tuberculosis was diagnosed in 59%.

In group 2 (breath sounds unchanged) 45 cases, clinical pulmonary tuberculosis was diagnosed in 60%.

In group 3 (breathing distant or exaggerated) 13 cases, clinical pulmonary tuberculosis was diagnosed in 67%.

In group 4 (rales indefinite) 36 cases, clinical pulmonary tuberculosis was diagnosed in 72%.

In group 5 (rales not in upper one-third) 93 cases, clinical pulmonary tuberculosis was diagnosed in 78%.

In group 6 (fine rales in upper one-third) 140 cases, clinical pulmonary tuberculosis was diagnosed in 82%.

In group 7 (moderately coarse rales in upper one-third) 425 cases, clinical pulmonary tuberculosis was diagnosed in 99%.

From this analysis it is readily seen that as far as diagnosis is concerned, the standards of interpretation used by me were only a little more than one-half efficient when only broncho vesicular breathing was heard. The finding of broncho vesicular breathing interpreted as abnormal was no more likely to be associated with clinical

pulmonary tuberculosis than when no breath changes were heard, providing, of course rales were not heard in either instances.

Distant or exaggerated breathing occurring alone was accompanied by a larger per cent of positive diagnosis than when either of the above occurred. It is only when rales appear that it can be said that diagnosis reaches such a per cent of positives as to make their finding relatively consistently valuable. This is demonstrated in groups 6 and 7. The moderately coarse rales, in upper one-third, from this analysis being the most valuable is diagnosis inasmuch as nearly all cases in which they occurred were diagnosed as having clincal pulmonary tuberculosis.

Definite parenchymatous x-ray changes were demonstrated in various groups as follows:

Group 1—(broncho-vesicular breathing) 40%.

Group 2—(unchanged breath sounds) 51%.

Group 3—(distant or exaggerated breathing) 31%.

Group 4—(indefinite rales) 53%.

Group 5—(fine or moderately coarse rales not in upper one-third) 67%.

Group 6—(fine rales in upper one-third) 78%.

Group 7—(moderately coarse rales in upper one-third) 98%.

Here it is also clearly shown that broncho vesicular breathing, as interpreted by me, is by itself not a reliable guide to the presence of parenchymatous lung changes. It is also clearly shown that one-half of those having no abnormal breath sounds nor rales could or did have parenchymatous lung changes. Thus making a negative lung examination by physical signs practically of no value. When rales occur, however, I can be quite certain of the existence of parenchymatous lung changes in the x-ray films and particularly so when moderately coarse rales are found in the upper one-third.

Tubercle bacilli were found at the sanatorium as follows:

Group 1—(broncho-vesicular breathing) 18%.

Group 2—(breath sounds unchanged) 22%.

Group 3—(distant or exaggerated breathing) 0%.

Group 4—(indefinite rales) 19%.

Group 5—(rales not in upper one-third) 43%.

Group 6—(fine rales in upper one-third) 45%.

Group 7—(moderately coarse rales in upper one-third) 71%.

From the above it can be said that when moderately coarse rales are heard the lesion is probably an open one.

Of course it can easily be said that all the cases in these analyses presumptively had clinical pulmonary tuberculosis and do not represent the occurrence of broncho-vesicular breathing interpreted as abnormal in the ordinary run of cases undergoing chest examinations. Nevertheless, only 59% were diagnosed as having the disease. A much more important criticism is that the standards for examination and interpretation were faulty. Undoubtedly they were. Can one, however, make the standard so exact as to be more valuable? Does my standard fall far below the average? I do not think so.

It is likewise true that most of the cases in which I found abnormal broncho-vesicular breathing had this change at the upper part of the right lung. But isn't it also true that here pulmonary tuberculosis is most likely to first show itself? How differentiate between the slightly abnormal broncho-vesicular breathing heard over diseased areas and the normal broncho-vesicular breathing due to anatomical structure? Rales permit of only a much narrower margin of interpretation and signify some pathologic change. In my experience at least they are the only auscultatory physical sign of any importance in the diagnosis of pulmonary tuberculosis unless breath sound changes are so marked as to approach the bronchial type.

Summary: From an analysis of cases presenting

1. Broncho-vesicular breathing interpreted as abnormal and without rales.
2. Unchanged breath sounds with no rales,
3. Distant or exaggerated breathing with no rales,
4. Indefinite rales,
5. Fine or moderately coarse rales elsewhere than in the upper one-third (breath sounds disregard),
6. Fine rales in the upper one-third (breath sounds disregard),
7. Moderately coarse rales in the upper one-third, (breath sounds disregarded),

it was found that

1. Moderate broncho-vesicular breathing in the absence of rales is not a safe sign upon which to base a diagnosis of clinical pulmonary tuberculosis. The chance of error is approximately 50%. Furthermore, less than one-half such cases have definite parenchymatous x-ray changes.

2. Unchanged breath sounds without rales may occur, although clinical pulmonary tuberculosis is present. In this series of selected cases 60% were diagnosed as having clinical pulmonary tuberculosis and about one-half showed definite parenchymatous x-ray changes

3. Distant or exaggerated breathing without rales was accompanied by a diagnosis of clinical pulmonary tuberculosis in 67%, although only 31% showed definite parenchymatous x-ray changes. Most of these few cases (13) had thickened pleura or pleurisy with effusion.

4. When rales occur, however, some definite pulmonary pathological change is most likely to be present. Regardless of breath sounds, when rales were heard clinical pulmonary tuberculosis was diagnosed in 78%—99% of the cases depending upon the location and type of rale heard. The x-ray showed definite pulmonary parenchymatous change in 67% to 98% also depending upon location and type of rale.

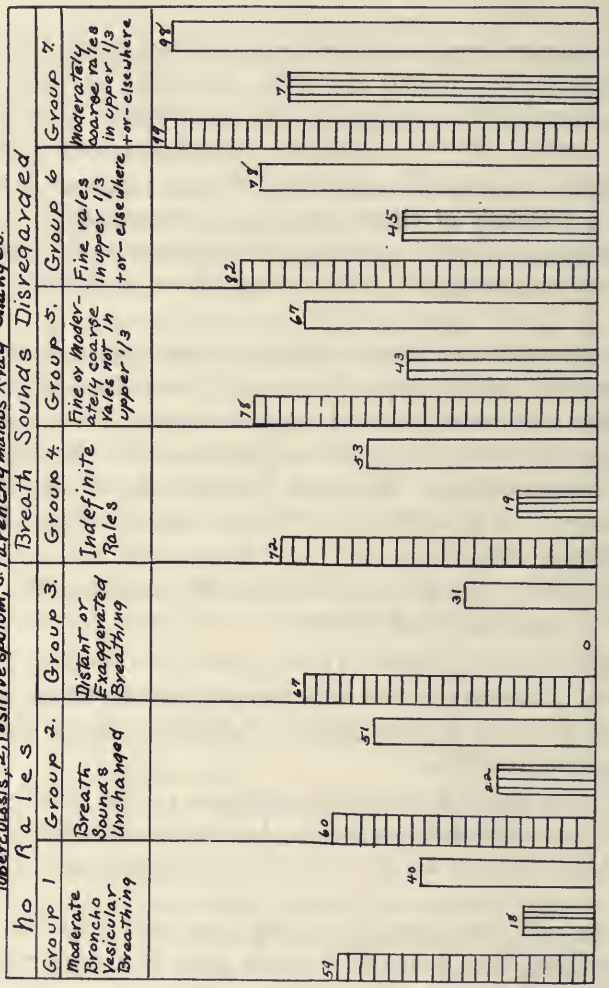
Conclusions: Using my own standards of interpretration and method of examination I have found:

1. That moderate changes in breath sounds are too often found when clinical pulmonary tuberculosis cannot be diagnosed to be of value in the diagnosis of this disease. Particularly is this true of broncho-vesicular breathing.

2. That a negative physical examination of the chest by no means rules out clinical tuberculosis. A negative examination has about the same value as the occurrence of broncho-vesicular breathing.

3. That rales heard especially in the upper portion of the lung —and particularly the moderately coarse rale—is a very reliable diagnostic sign of clinical pulmonary tuberculosis. As a matter of fact, it is the only reliable auscultatory sign unless the changes in breathing are very marked. It is not to be inferred from this, how-

ever, that whenever rales are heard clinical pulmonary tuberculosis is always present.



DISCUSSION.

Dr. Perkins: I wish to impress the fact that these are sanatorium patients on whom a diagnosis has already been made. If we attempted the diagnosis of tuberculosis in accordance with these percentages here, we should fill the sanatoriums several times over. We are finding those signs often. Particularly following the influenza epidemic, we found many patients giving that sign, a very small percentage of whom have tuberculosis.

Dr. Otis: The differential diagnosis between a chronic extensive case of bronchiectasis and pulmonary tuberculosis is not infrequently exceedingly difficult to make. Both the symptoms, physical signs and even the x-ray picture are sometimes so much alike in both diseases. Of course in bronchiectasis the tubercle bacilli are always absent and the lesion is usually in the base of the lungs. I recall the case of a girl of twelve years who came to my clinic and who, during nine years out of the twelve, was treated either in a sanatorium or consumptive hospital as a far advanced case of tuberculosis. I believe the case was one of bronchiectasis, and she had been treated all these years under the mistaken diagnosis of far-advanced tuberculosis. I recall another case which was referred to me as one of tuberculosis with a cavity in one of the upper lobes. Both from the characteristic symptoms of bronchiectasis and the absence of tubercle bacilli I felt sure that this was a case of long-standing bronchiectasis and not one of tuberculosis. Pulmonary tuberculosis is bad enough, but a chronic bronchiectasis is worse and offers less prospect of relief.

Dr. Wilson: The report of a couple of cases referred to by Madison of Milwaukee, says that rales were found over a period of several years, but on autopsy no evidence of tuberculosis could be found. I have had a group who I have followed for years, giving tuberculin to two patients, and yet these rales persist, but no tuberculosis has been found. In most of the reports and discussions of cases of this kind, the diagnosis seems to depend entirely upon the rales.

Dr. Otis: Slight changes in the percussion note and in the respiratory murmur are uncertain and dangerous grounds upon which to make a definite diagnosis of pulmonary tuberculosis. The persistent apex rale is and always will be the most important and definite sign of existing disease, and I am glad that Dr. Heise has emphasized the extreme value of this sign. It often requires skill, experience, and practice, however, to elicit the apex rale.

Dr. Probst: I should like to ask the speaker, what is the significance of rales when they continue for years in a patient who is apparently entirely well? I have had patients under observation for six or eight years or more with persistent rales in the same location, who are seemingly in perfect health.

Dr. Hawes: I sometimes think that students make a fetish out of rales. If they find rales the person has tuberculosis; if they do not find rales, even if the man is at death's door, he has no tuberculosis. They do not depend enough on the appearance of the man himself nor on the evidence obtained from a careful history.

Dr. Heise: I have found, 25 years after their discharge, rales in the same area as when they were patients, the patients remaining perfectly well in the meantime. A similar group that were apparently arrested without rales do better in a long run and the short run, than the bacilli negative. The rales must be looked at as potential breakdown.

In regard to Dr. Murray's question, I might say that for years, six years at least, we controlled x-ray examination by fluoroscopic methods, and then when we were satisfied that they were worthless, we discarded them. We do not take oblique pictures in those cases where we do not find definite tuberculosis in the lung when the patient has tubercle bacilli.

I think Dr. Hawes' remarks are well taken. I very sincerely question the tuberculous nature of any rales in the chest, if you cannot substantiate a change in the lung by means of an x-ray. I certainly believe that any tuberculosis that will give you rales in the lungs, will show a definite change in the x-ray picture.

In answer to Dr. Wilson: We do get cases in which we do not find tuberculosis but do find rales.

TUBERCULOSIS MORTALITY IN RELATION TO ALTITUDE, HUMIDITY AND POPULATION DENSITY.

By C. F. GARDINER, M. D., G. B. WEBB, M. D., AND C. T. RYDER, M. D.

The relation between climate and tuberculosis is a question which has intrigued physicians since Hippocrates, and no doubt savages and their medicine men have puzzled over it since the dawn of thought, or since man's first encounter with the disease. Nevertheless it still remains in the foggy regions of speculation and prejudice. The available data are so inadequate and present such complex difficulties of interpretation that it has been the natural thing to ignore them and to form opinions untrammelled by any attempt to learn the facts.

Ichok, in a recent study[1], has tried, with full recognition of the perplexities involved, to put the problem on a rational basis. He divides France into seven climatic regions which are described with references to temperature, humidity, rainfall, altitude, and wind, and compares their tuberculosis mortality, general, urban (towns of more than 5000 inhabitants), and rural. Allowing for possible sources of fallacy, he concludes that climatic factors have an influence on tuberculosis motality, though this influence is only relative, not absolute; that "the climate of Auvergne takes first place (with the lowest mortality) which is to be attributed to the altitude of the region"; that "the bad effects correlated with humidity and frequent rains are apparent in the climatic regions of Brittany and Paris" (rural as well as urban).

We have undertaken to make a similar study for the United States, making use of the data provided by the United States Census Bureau, Weather Bureau, and Geographic Survey. We tabulate these data by states for which mortality figures are available, showing the approximate mean altitude, humidity (when available), density of poulation, and death rates from tuberculosis (Table 1). The figures on humidity are approximate averages taken from several

stations over a period of years, and usually based on observations made at 8 A. M. and 8 P. M. The word approximate should be stressed, for the data provided by the Weather Bureau on this important point have not been adequate in the past; at present they are being rapidly and greatly improved. Future reports will cover drying and cooling rates, which are the resultant of relative humidity and air movement, and have been shown by Leonard Hill to bear an important relation to metabolism. The figures on population density are from the 1920 census and those on death rates from the 1920 mortality statistics. It did not seem possible to take averages over longer periods, as a number of states have only recently entered the registration area; and while death rates fluctuate, the relation of state to state in this respect remains fairly constant.

Before attempting to correlate, even in the roughest way, the tuberculosis death rates with any of the factors considered, it is necessary to analyze this table and make some exclusions from it. As regards states with a very large negro population, the Report of the Census Bureau calls attention to the very much higher mortality from tuberculosis in this race than among whites, and cautions against making regional comparisons without allowing for this; we therefore exclude the Southern states. The same report says, "There are many localities to which persons afflicted with this disease (tuberculosis) are attracted because of climatic conditions favorable to the treatment of this disease. States and cities which annually show high death rates from tuberculosis (all forms) are not always those which have conditions tending to the development of tuberculous disease, but often are health resorts." On the strength of this obvious fact we have excluded Colorado and California, the registration states having the largest number of imported tuberculous invalids.

It will be noted that the death rate from tuberculosis (all forms) in Colorado in 1920 was 225.4. A recent study by Sewall[2] indicates that less than 17% of these deaths are among native Coloradoans. The rate for Denver in 1920 was 318.1 per 100,000 but the deaths from tuberculosis "contracted in Colorado", as determined by the Denver Department of Health, were less than 16% of the total. In 1905 one of the present writers (C. F. G.), who had been studying

the matter closely since 1889, wrote, "At Colorado Springs, with a population of from 15,000 to 30,000, there has been but one (fatal) case each year originationg among the native population..... I have no record of other forms than pulmonary, but meningeal, glandular, or bone tuberculosis is not more frequent than in other places, although a large proportion of our children have a tubercular parentage"[3]. During the past 11 years the deaths from non-imported pulmonary tuberculosis have averaged 2.1 per year in a population of about 30,000, possibly half of which is made up of health seekers and their families. Even if we assume that only 10,000 are natives and therefore candidates for tuberculosis "contracted in Colorado" this still gives a rate of only about 20 per 100,000, or about 1/7 of the rate for the country at large during the same period. Such data however are open to more criticism than the rest of the material with which we are dealing, and we have no similar reports from California, so we have thought best to omit these two resort states from the graphic correlations.

For the remaining group of 22 states*, we have prepared diagrams showing the death rates per 100,000 from tuberculosis (all forms) in relation to altitude, humidity, and density of population (figures 1, 2, 3). It is clear that many possible sources of misinterpretation still remain A state with a very small population per square mile may have a large percentage of that population crowded into one or two cities. A state with a high mean altitude may have most of its population living along the coast practically at sea level. A dry state may report a rather high relative humidity because the stations are located in the more humid sections, or vice versa. We do not mean to draw any dogmatic conclusions from material presented, but merely to offer it, following Ichok's good example, as a contribution to the study of climate in its relation to disease.

With this insistent disclaimer of any conclusive certainty, we may note some inferences suggested by the curves. In general, the states showing high mortality are those which combine low altitude, high humidity, and marked density of population,—the industrial

*Twenty-one states in the case of humidity. No figures for New Hampshire.

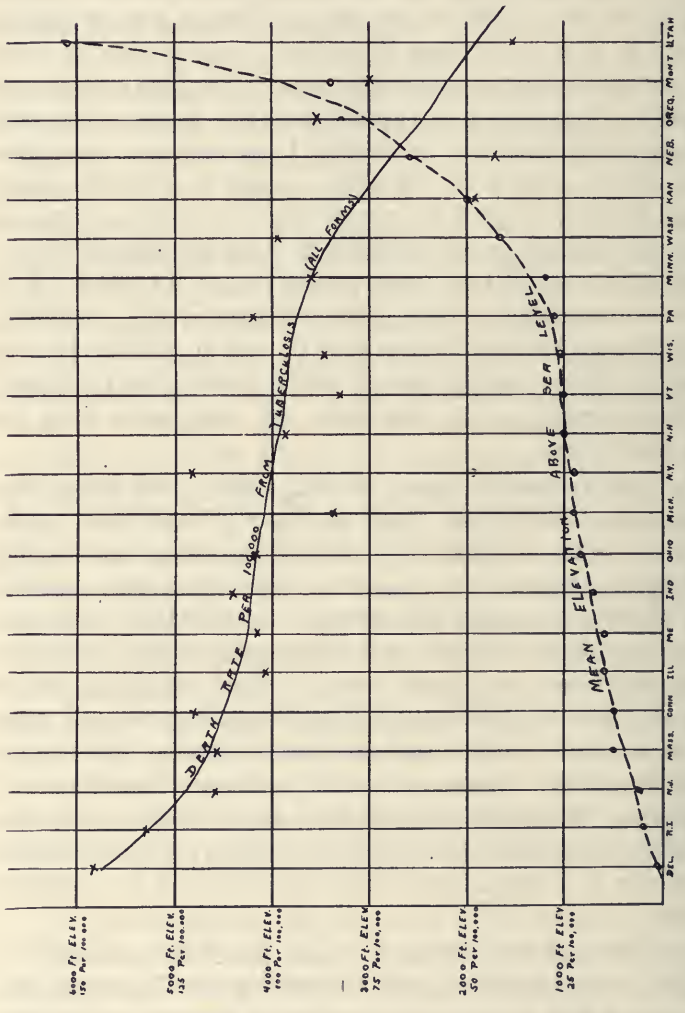

6000 Ft ELEV.
150 PER 100,000
5000 Ft. ELEV.
125 PER 100,000
4000 Ft. ELEV.
100 PER 100,000
3000 Ft. ELEV
75 PER 100,000
2000 Ft. ELEV
50 PER 100,000
1000 Ft ELEV
25 PER 100,000
DEATH RATE PER 100,000 FROM TUBERCULOSIS
(ALL FORMS)
MEAN ELEVATION ABOVE SEA LEVEL
DEL. N.J. Md. MASS. CONN ILL. PA. IND OHIO MICH. N.Y. N.H VT WIS. PA MINN. WASH KAN NEB. OREG. MONT UTAH

Fig 1.

Curves of Altitude and Tuberculosis Mortality Plotted Together

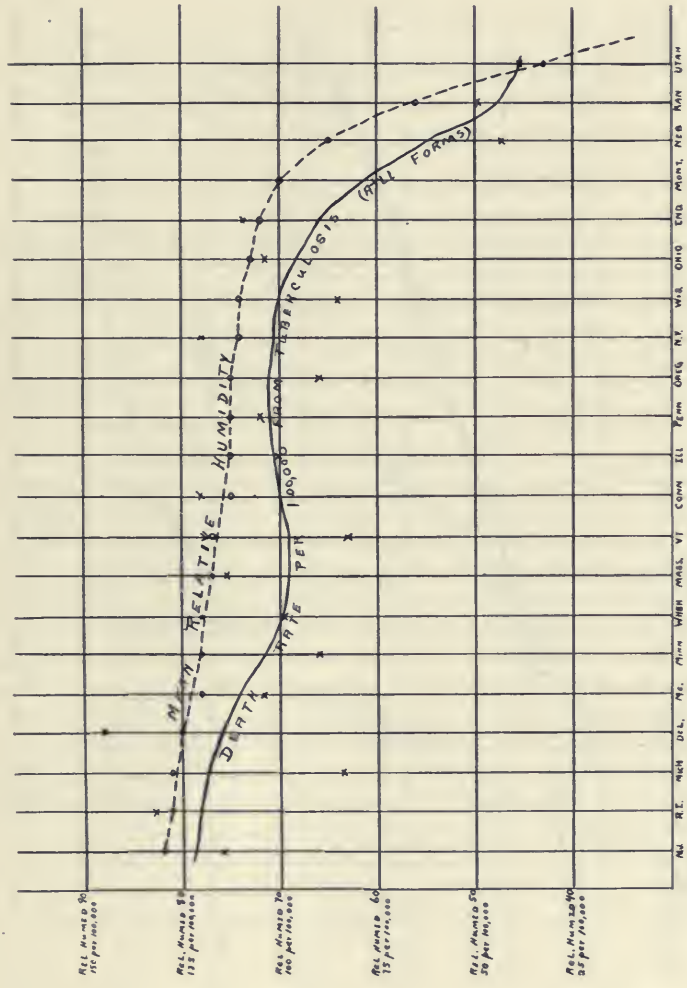

MEAN RELATIVE HUMIDITY
DEATH RATE PER 100,000 FROM TUBERCULOSIS (ALL FORMS)
Rel. Humid 90 155 per 100,000
Rel. Humid 80 130 per 100,000
Rel. Humid 70 100 per 100,000
Rel. Humid 60 75 per 100,000
Rel. Humid 50 50 per 100,000
Rel. Humid 40 25 per 100,000
Ala. Ariz. Ark. Cal. Colo. Conn. Del. Fla. Ga. Ida. Ill. Ind. Ia. Kan. Ky. La. Me. Md. Mass. Mich. Minn. Miss. Mo. Mont. Neb. Nev. N.H. N.J. N.M. N.Y. N.C. N.D. Ohio Okla. Ore. Pa. R.I. S.C. S.D. Tenn. Tex. Utah Vt. Va. Wash. W.Va. Wis. Wyo.

FIG 2

Curves of Relative Humidity and Tuberculosis Mortality Plotted Together.

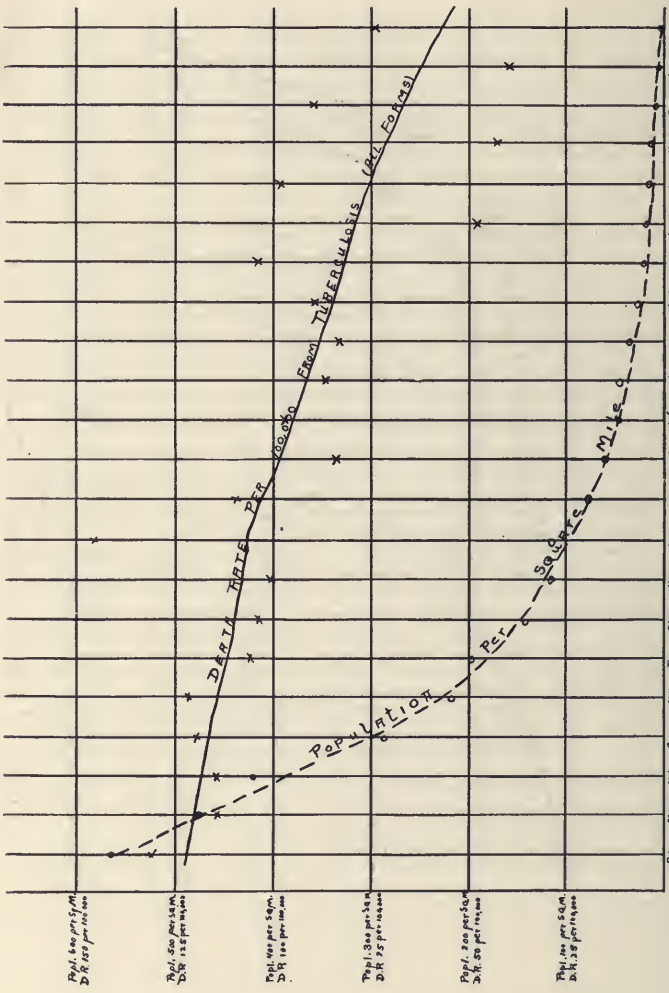

DEATH RATE PER 100,000 FROM TUBERCULOSIS (ALL FORMS)
POPULATION PER SQUARE MILE
R.I. Mass. N.J. Conn. N.Y. Pa. Ohio Ill. Del. Ind. Mich. N.H. Wis. Vt. Minn. Me. Kan. Wash. Neb. Ores. Utah Mont.
Popl. 600 per Sq.M. D.R. 150 per 100,000
Popl. 500 per Sq.M. D.R. 125 per 100,000
Popl. 400 per Sq.M. D.R. 100 per 100,000
Popl. 300 per Sq.M. D.R. 75 per 100,000
Popl. 200 per Sq.M. D.R. 50 per 100,000
Popl. 100 per Sq.M. D.R. 25 per 100,000

Fig. 3
Curves of Population Density and Tuberculosis Mortality Plotted Together

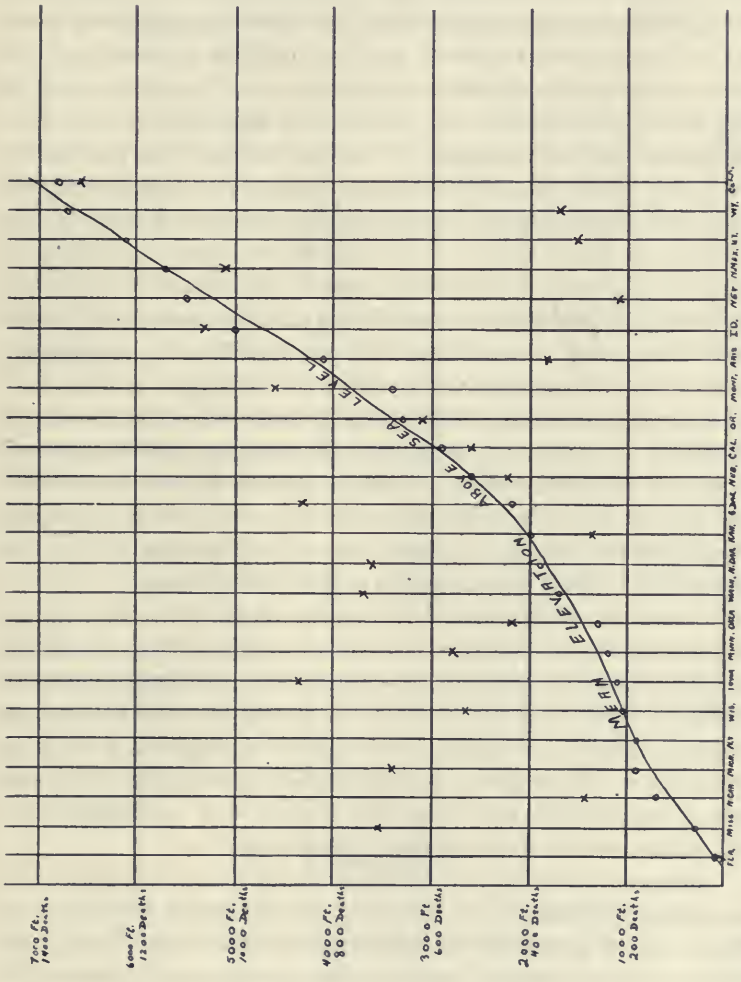

7000 Ft. 1400 Deaths.
6000 Ft. 1200 Deaths.
5000 Ft. 1000 Deaths.
4000 Ft. 800 Deaths.
3000 Ft. 600 Deaths.
2000 Ft. 400 Deaths.
1000 Ft. 200 Deaths.
MEAN ELEVATION
ABOVE SEA LEVEL
U.S. Mtns. Mean Mean Alt. Wis. Iowa Minn. Ohio Mass. Mean Alt. Ky. Ind. Ill. Mich. Ark. Kans. Neb. Cal. Ore. Mont. Ariz. Ida. Nev. N.Mex. Wy. Colo.

Fig. 4. Altitude and Indian Tuberculosis Mortality Plotted Together

coast states; while the states showing decidedly low death rates are high, dry, and sparsely populated—the inland plateau states. The average rates for the latter region are half or less than half those of the former. Whether we arrange the states in order of ascending altitude, decreasing humidity, or decreasing population density, the same group of states occupies the first division, and the same group the last division; and the curve of mortality drawn in relation to each of these other curves as a basis falls in a more or less similar way on each chart. However they are not exactly the same, and inspection, verified by a rough mathematical analysis, shows that the correlation between death rates and climatic factors is somewhat closer than that between death rates and population densities. Moreover the low death rates of the favored states (about 40 per 100,000) are not only much lower than the general rate for the registration states as a whole (113.2 per 100,000), but much lower than the rural rate for that area (108.1 per 100,000).

From these facts we think it fair to infer that while scarcity of population, character of population and industries, relative absence of poverty, and so forth, are of great importance, there are in addition climatic factors, notably altitude and dryness, which are largely accountable for the extraordinarily low tuberculosis death rates prevailing in the inland plateau region of the United States.

In addition to the general tuberculosis death rates, we have for our own satisfaction plotted the curves of mortality from pulmonary and non-pulmonary tuberculosis in relation to the same factors. In general the three mortality curves ran closely parallel, and it does not seem desirable to multiply or complicate diagrams in order to show them. The relation between descending mortality from pulmonary tuberculosis and ascending altitude was somewhat closer than in the case of non-pulmonary tuberculosis.

A recent report by a Committee of the National Tuberculosis Association appointed to collaborate with the Senate Committee on Indian Affairs[4] provides data which enable us to make some crude comparisons between the regional tuberculosis death rates in this race and in the population at large. Feeling that this might add something to the valuable studies of Bushnell[5], Cummins[6], Deycke and Much[7], and others, on the differences between tuberculosis in

races which have long been accustomed to it and races to whom the disease is new, we analyzed these data in the same way. (Table 2). Relative density of Indian population could not be determined. As regards altitude and dryness, the correlation with tuberculosis death rates which appeared to exist in the case of the comparatively immune general population is entirely wanting in the case of the susceptible savage. It was impossible to draw a resultant curve of mortality rates, which were scattered helter-skelter all over the chart (Figure 4). There seemed to be greater susceptibility in those Indians living in the newest parts of the country and hence having the shortest racial history of exposure, but this was not constant enough to be stated as a fact.

It is of course true in general that the more immunity exists already, the more it can be exalted by favorable conditions to which an organism of low resistance will not respond. This general statement probably applies in the case of the Indians. However it has been reported that in South America, the Indians of the high Andes show much lower tuberculosis death rates than those of the plains[8]. As regards another class of highly susceptible material, namely experimental animals, we noted some years ago[9] that rabbits inoculated with bovine tubercle bacilli seemed to live longer at Colorado Springs (6,000 ft.) than at sea level, and we have recently observed the same thing in the case of guinea pigs inoculated with human tubercle bacilli, though the differences are neither great nor constant. The number of lymphocytes[10] and mononuclear leucocytes in the blood is as shown first by Webb and Williams, increased in these animals, as in man, at high altitudes, and we believe this fact to have a bearing—much discussed but not yet explained—on resistance to tuberculosis.

Conclusion.

The high inland plateau region of the United States has a remarkably low death rate from tuberculosis, both pulmonary and non-pulmonary, as compared with lower and damper regions, and this appears to be due not only to favorable hygenic, economic, and social conditions, but also in considerable part to climatic factors, notably altitude and dryness.

REFERENCES

1. Ichok. Revue de la Tuberc., III, 3, 1922, p. 267.
2. Sewall. Address to Denver Sanatorium Society, April 24, 1923.
3. Gardiner. Trans. Nat. Tuberc. Assn., 1905, p. 316.
4. Tuberculosis among the North American Indians. Senate Committee Print, 1923.
5. Bushnell. Epidemiology of Tuberculosis. 1920.
6. Cummins. Brit. J. Tuberc., Oct., 1920, p. 160.
7. Much. Tuberculosis of Children. American edition, 1921.
8. Evans. Phthisiology, Historical and Geographical. 1888.
9. Webb, Williams, and Basinger. Trans. Nat. Tuberc. Assn., 1910, p. 279.
10. Webb and Williams. The Increase of Lymphaocytes at High Altitudes. National Tuberculosis Assn. Transactions. 1911.

State	Approx. Mean Elevation In feet	Approx. Mean Relative Humidity	Population per Square Mile	Death Rates per 100,000 Inhab. All Forms TB.	Pulmonary TB.	Non-Pulmonary TB.
Delaware	60	80	113.5	146.0	131.7	14.3
Florida	100	80	17.7	104.1	95.1	9.0
white				67.1	59.7	7.4
colored				176.8	164.4	12.4
Louisiana	100	76	39.6	141.2	132.7	8.5
white				80.9	74.4	6.5
colored				236.2	224.5	11.7
Rhode Island	200	81	566.4	131.3	110.6	20.7
New Jersey	250	82	420.0	114.0	102.0	12.0
Mississippi	300	78	38.6	127.8	121.2	6.6
white				47.8	44.7	3.1
colored				201.2	191.5	9.7
South Carolina	350		55.2	120.0	111.9	8.1
white				65.5	58.9	6.6
colored				172.0	162.5	9.5
Maryland	350	74	145.8	146.8	132.1	14.7
white				110.4	99.7	10.7
colored				326.6	292.0	34.6
Massachusetts	500	77	479.2	113.8	96.9	16.9
Connecticut	500	75	286.4	119.4	103.8	15.6
Illinois	600	75	115.7	100.6	89.1	11.5
Maine	600	78	25.7	103.8	85.1	18.7
Indiana	700	72	81.3	108.8	92.9	15.9
North Carolina	700	84	52.5	116.5	106.4	10.1
white				82.8	75.3	7.5
colored				194.2	178.2	16.0

State	Approx. Mean Elevation In feet	Approx. Mean Relative Humidity	Population per Square Mile	Death Rates per 100,00 Inhab. All Forms TB.	Pulmonary TB.	Non-Pulmonary TB.
Kentucky	750	76	60.1	152.9	133.9	19.0
Missouri	800	71	49.5	106.9	97.9	9.0
Ohio	850	73	141.4	102.8	88.3	14.5
Michigan	900	81	63.8	83.6	72.0	11.6
New York	900	74	217.9	120.9	106.1	14.8
Tennessee	900	76	56.1	164.6	145.2	19.4
white				129.3	112.8	16.5
colored				312.6	281.1	31.5
Virginia	950	79	57.4	142.9	128.9	14.0
white				94.8	84.1	10.7
colored				256.2	234.6	21.6
New Hampshire	1000		49.1	97.0	81.4	15.6
Vermont	1000	77	38.6	81.8	67.0	14.8
Wisconsin	1050	74	47.6	85.6	75.0	11.6
Pennsylvania	1100	75	194.5	105.0	91.5	13.5
Minnesota	1200	78	29.5	89.6	77.1	12.5
Washington	1700	78	20.3	98.4	81.5	16.9
Kansas	2000	56	21.6	48.2	41.2	7.0
U. S. A. (1)	2500		35.5	113.2	99.8	13.4
Nebraska	2600	65	16.9	43.0	35.4	7.6
California (2)	2900	77	22.0	159.6	141.8	17.8
Oregon	3300	75	8.2	89.1	73.2	15.9
Montana	3400	70	3.8	75.1	65.6	9.5
Utah	6100	43	5.5	39.1	32.0	7.1
Colorado (2)	6800	45	9.1	225.4	212.5	12.9

(1) Registration States.

(2) Includes great numbers of imported cases: see text.

State	Approx. Mean Elevation In Feet	Approx. Mean Relative Humidity	Indian Population	Indian Deaths From Tuberculosis	Indian TB. Mortality per 100,00
Florida	100	80	454	0	0
Mississippi	300	78	1400	10	714
North Carolina	700	84	2432	6	248
Michigan	900	81	1093	7	640
New York	900	74	6072		
Wisconsin	1050	74	9713	52	535
Iowa	1100	70	345	3	870
Minnesota	1200	78	12378	69	558
Oklahoma	1300	70	15028	66	439
Washington	1700	78	9639	71	736
North Dakota	1900	71	9018	65	721
Kansas	2000	56	1466	4	272
South Dakota	2200	70	22649	197	870
U. S. A.			336337		446
Nebraska	2600	65	2461	11	447
California	2900	77	13241	69	521
Oregon	3300	75	4042	25	618
Montana	3400	70	12374	114	921
Arizona	4100	38	42400	153	361
Idaho	5000	63	4048	43	1062
Nevada	5500	50	10900	23	211
New Mexico	5700	45	21530	220	1022
Utah	6100	43	1641	5	305
Wyoming	6700	57	1748	6	343
Colorado	6800	45	796	11	1382

DISCUSSION.

DR. J. M. MILLER: To draw any conclusions from statistics, it would seem that there are other factors that were more important. There are certainly a great many factors that are more important in the incidence of tuberculosis than are these factors of environment.

DR. WEBB: I do not lay great stress on this paper, but just presented it as a short contribution.

DIAGNOSIS OF LUNG INFECTIONS IN CHILDREN.

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The diagnosis of acute respiratory infections is at times very difficult and I feel quite sure that many cases of prolonged acute bronchitis are in reality cases of unrecognized broncho-pneumonia. However, careful observation of the progress of the case in most instances clears up the diagnosis after a few days.

Quite a different problem is met in the chronic pulmonary infections in children under twelve years of age, and it is with this group that this paper will deal.

Many children admitted to the Monroe County Sanatorium with a diagnosis of tuberculosis have been found to have some other infection as the cause of the illness. Other children have been treated for non-tuberculous lung infection many months before the true state of affairs was discovered and referred for treatment.

The following fourteen cases illustrate the point I wish to make, viz: If in doubt play safe and treat the condition as tuberculosis until the case may be proven to be something else.

Case 1.—M—Age 12—Admitted 10/19/'20. Mother died tbc. 1912. Has been under treatment in M. C. T. S. three previous admissions. Cough severe—muco purulent sputum one to 3 ozs. daily, persistently neg. for tbc.—Wasserman negative—O. T. positive. Temp. and pulse slightly elevated—Under weight. Lungs—many musical and large and medium moist rales lower 2/3 left. X-ray showed heavy trunks in both bases. During residence had several acute febrile attacks—probably broncho-pneumonia. Lungs now show many moist sounds lower 2/3 and x-ray shows shadings suggestive of Bronchiectasis. Diagnosis: Chronic Bronchitis—Bronchiectasis.

Case 2.—M—Age 9—Admitted to Sanatorium 3/4/19. Mother died tuberculosis 1917. Pneumonia 1915 followed by Empyema and drainage. Diagnosed as tuberculous and sent to Sana-

torium. Under weight—T. 99.8—P. 100—No cough—Wassermann negative. Tonsils 2 plus—O. T. Negative. Lungs—left shows old op. scar post auxiliary line—increased whispered voice and rales left base back. Recurrence empyema 11/17/19—drainage op.—Staph & streps—No tbc. Discharged 8/4/20—Sinus closed—general condition good. Diagnosis: Empyema.

Case 3.—M—Age 7—Admitted 11/5/21. During 1921 had chicken pox, scarlet fever, double otitis media and pneumonia followed by right empyema operation before admission. Cough severe—raised heavy pus in moderate amount. Left ear discharging—Empyema wound still draining. Lungs on adm.—area in nipple region right suggesting fluid or consolidation around op. scar. Both lungs showed rales throughout after cough. Fingers showed marked clubbing. Sputum neg. for tbc.—Urine and Wassermann negative. O. T. Positive. Smear from left ear positive for tbc. and diphtheria. X-ray on admission shows hazy shadow lower ½ right and lower 2/3 left. After rest in bed temperature became normal and general condition improved. Cough and sputum continued but prolonged postural drainage reduced both markedly. X-ray later showed shadows suggesting cavities in right base and dense shadow upper ½ left toward axilla suggesting fluid or thickened pleaura. Later lungs were much clearer on x-ray and patient discharged much improved. Diagnosis: Empyema—Pneumonia—Bronchietasis—Tbc. Otitis Media.

Case 4.—13 months old. January 1923 had pneumonia with consolidation in lower right. In eight days temperature became normal. Three days later had another rise of temperature, loss of appetite with marked cough, dyspnoea, etc. Temperature continued elevated, child apparently growing worse slowly, until seen February 24th. At this time signs of fluid were obtained low in the right back near the spine. Child was diagnosed as Empyema and sent to hospital for further study and treatment. X-ray on admission showed a dense shadow extending from the heart shadow on the right side at base. An attempt was made to aspirate for diagnosis, with the result that the entire lung collapsed and remained collapsed for three or four weeks. During this time temperature became normal and general condition improved rapidly. Patient was discharged

from hospital April 21, in apparently normal condition. Diagnosis: What?

Case 5.—Female—Age 12—Admitted 3/16/22. Tonsil and Adenoid operation—Ether—one year ago—developed pulmonary abscess. Cough continued after abscess ruptured and she did not regain strength. Seen and examined 2/12/22.—Signs of lesion above 3rd and 4th v. s.—increased whisper prolonged expiration and rales. O. T. positive. X-ray shows abscess with suspicious markings above to apex. Discharged 10/20/22 doubtful tuberculous with no signs in chest and x-ray practically clear.

Case 6.—F. B.—Age 11. From age of three had frequent asthmatic seizures. In the winter of 1919 had pneumonia of right lower lobe. The signs of pneumonia did not clear up, although temperature and pulse came to normal and general condition improved greatly. Was seen in consultation in fall of 1920. At this time signs in right chest were those of a resolving pneumonia, with the absence of any dullness on percussion and bronchial breathing. Asthmatic seizures continued at frequent intervals and during the winter months he was almost constantly short of breath and coughing. He was found to be sensitized to almonds and cat dandruft. Was sent south for three winters, each time being practicaly free of asthma while in the warm climate, but each time returned to Rochester in bad weather and blew up with asthma almost immediately upon return. Was seen again in fall of 1922, during an asthmatic seizure and specimen was obtained from his larynx, which sputum showed positive for tubercle bacilli. Spent last winter in Asheville, was in bed most of the time, general condition and weight improved markedly, but signs in chest remain almost unchanged. Diagnosis: What?

Case 7.—Female—Age 12—Admitted 5/23/19. Epilepsy up to six years of age—no seizure since. Bronchitis at one year of age —has coughed since. On admission showed numerous rales through lower half both chests. Tonsils 2 plus—O. T. positive. X-ray showed increase in hilum shadow—descending trunks thickened. Large quantity negative sputum. Diagnosed as Bronchiectasis. Improved under rest and postural drainage. Sputum found positive 12/22/22. Diagnosis: Bronchiectasis—Pulmonary tbc.

Case 8.—Female—Age 5—Admitted 4/27/20. Mother died

tuberculosis April 1921. No history of illness—examined as contact case. Child is undernourished—has poor appetite and temperature is elevated to 100 in afternoon. X-ray shows hilum enlarged on left; probably on right as well. O. T. Pos. Physical examination did not show any signs of disease. During residence she has had frequent slight elevation of temperature and pulse but has improved steadily in weight and general condition. An attack of lobar pneumonia intervened in April 1922 without any flare-up in tuberculous process. Diagnosis: Hilum tuberculosis.

Case 9.—M—Age 6—Admitted 9/7/22. Father had tuberculosis and died in Sanatorium in November 1922. Examined as contact case—was under weight, listless, had moderate cough and temperature slightly elevated. Chest showed increased whisper down spine to 8th v. s. with broncho-vesicular breathing inside angle right scapula. X-ray showed dense shadow to right of hilum. O. T. positive—No Sputum—Wassermann negative. Present condition very satisfactory. Diagnosis: Hilum tuberculosis.

Case 10.—Female—Age 10—Admitted 8/2/22. Family history negative. Patient was kept from school several weeks past three winters on account cough. In June 1922 was always tired and had pain in right lower abdomen. Operated 6/8/22 for acute appendicitis when tuberculous peritonitis was found. Patient developed cough and was sent to Sanatorium. O. T. positive. Few fine rales both bases. X-ray suggested Hilum tuberculosis. Diagnosis: Tuberculous peritonitis—hilum tuberculosis?

Case 11.—Male—Age 9—Admitted to Sanatorium 12/17/18. Measles—Flu six weeks before admission. O. T. positive. Symptoms—marked cough and weakness, sweats, etc. Sputum showed many bacilli. Lungs—Signs advanced tuberculosis both sides. X-ray—marked involvement throughout both lungs. Died with large pulmonary hemorrhage 2/2/19. Diagnosis: Pulmonary tuberculosis.

Case 12.—Female—Age 7—Admitted to Sanatorium 6/4/18. Mother died in Sanatorium 1918—Also Maternal Aunt died 1920: Patient admitted account being run down and cervical adenitis—scars both sides of neck from former abscesses. O. T. positive. Lungs—negative on admission—X-ray negative—No sputum—Urine showed pus cells and much albumen. Child had done badly—Cer-

vical glands have enlarged and broken down and drained several times—Kidneys progressed—Lungs now show signs: diminished breath sounds and medium moist rales left lower lobe. Diagnosis: Pulmonary tuberculosis (?)—Tuberculous cervical adenitis—Tbc(?) Kidneys.

Case 13.—Female—Age 10—Admitted 4/22/19. Advanced tuberculosis—many tbc in sputum—O. T. positive. Sister has advanced tuberculosis in home. Case of interest on account of apical location of lesion. Disease progressed and patient died 8/2/20.

Case 14.—Male—Age 6—Admitted 2/15/19. Sputum contains bacilli—O. T. positive. Signs extensive involvement both lungs with X-ray suggesting miliary type. Died in hospital 6/24/19.

Case 15.—Male—Age 13—Admitted 9/19/19. Four weeks before admission taken sick with chills and high temperature, aggravated cough, etc. Attending physician aspirated small amount fluid from right pleural cavity. On admission patient showed pneumothorax of right side with compressed lung crowded downward. Foul pus aspirated from right pleural space several times. 9/30/19—Resection operation and drainage under gas oxygen anaesthesia—large amount pus removed. 10/10/19—Copious hemorrhage through empyema tube resulting in death. Autopsy showed pleural space filled with blood clot—lung partially collapsed toward base—lung showed multiple abscess cavities in a pneumonic lung. Diagnosis: Lung abscess—Empyema—Fatal hemorrhage.

Although case 15 was over twelve years of age it is inserted here because of the rather unique termination, and the interesting autopsy findings.

In practically all these cases an incorrect diagnosis was made at first whereas more careful study would have made possible a correct diagnosis earlier in many of the cases.

We are all aware of the rapidity with which a lung abscess burrows and extends at times and the consequent danger in delayed diagnosis.

Bronchiectasis is another condition that yields to treatment if instituted early in its course and is almost a hopeless condition so far as recovery is concerned if the delay be a long one. Surely delay is equally as serious in dealing with pulmonary tuberculosis.

I feel very strongly that the same general rule regarding the location of the lesion holds in children as in adults, viz: An apical lesion is tuberculous and a basal lesion non-tuberculous until proven otherwise. We have usually been unable to recognize a basal tuberculosis until bacilli have appeared in the sputum.

Tuberculosis of the hilum is a different diagnosis, and one that is unquestionably frequently overlooked. It would seem that we are warranted in reaching this diagnosis if we can obtain the following data: First—a history of contact with a known case of tuberculosis, which however, is not essential; second—a positive skin test; third —a sick child; fourth—an x-ray plate that may or may not show what we regard as suspicious markings; and fifth, no other demonstrable cause to explain the illness.

We know that childhood infection with the tubercle bacillus is very frequent and that the development of tuberculosis in the young adult is also not rare. Between these two extremes there must be innumerable periods of relative or actual activity in the potential disease focus which by proper care at the right time may be prevented from developing into serious activity in later years.

This can only be accomplished if we diagnose the condition when present, and this in the light of our present knowledge may be done only by a diagnosis reached by the process of exclusion.

CAUSES OF FAILURE IN THE TREATMENT OF PULMONARY TUBERCULOSIS BY ARTIFICIAL PNEUMOTHORAX

(Based on the Observation of Six Hundred Cases During a Period of Twelve Years)

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For the purpose of this study we have reviewed six hundred cases of pulmonary tuberculosis treated by artificial pneumothorax during the past twelve years, and have carefully analyzed every case terminating fatally to determine if possible the reason why a satisfactory end result was not obtained.

A correct determination of the causes of failure in each case presented many difficulties because most of our patients subjected to artificial pneumothorax were far advanced in their disease, usually with cavities on the worse diseased side while the contra-lateral lung was seldom entirely free from invasion, also complications of a serious nature, such as laryngitis and enteritis were often present.

Thus it is evident, for instance, that it would be difficult to say what was the primary cause of failure to obtain an "arrest," or "cure," in a chiefly unilateral cavernous tuberculosis case where adhesions prevented an entirely satisfactory collapse; and extension or progression of disease took place in the contralateral lung, later an enteritis and laryngitis developed, and the patient died from exhaustion, because any one or a combination of all of the above complications could be responsible for the failure.

Of course, there are other factors, many of them undeterminable, concerned in disturbing the state of balance between the invasive powers of the germ and the defensive powers of the body; however, we have critically studied each case and determined as accurately as possible the factor which we believe primarily respon-

sible for the failure of artificial pneumothorax to bring about a satisfactory outcome.

While many cases were of a desolate character and artificial pneumothorax was a last result procedure, most cases while advanced, were clinically of a type giving favorable results and it was our desire to secure information on this latter group particularly, which prompted this study.

For statistical purposes, our pneumothorax material has been classified into eight main groups as follows:

1. Severe acute infiltrating, caseous pneumonic and caseous Bronchopneumonic types of disease.
2. Chiefly unilateral fibro-caseous, progressive with little or no excavation.
3. Chiefly unilateral fibro-caseous cavernous progressive.
4. Chiefly unilateral fibro-caseous, cavernous; cavity manifestations in the foreground.
5. Tuberculosis pleuritis with effusion:—cortical fibro-caseous tuberculosis.
6. Severe hemoptysis.
7. Desolate bilateral cases. (Ultimum Refugium. Pneumothorax performed as therapeutic procedure.)
8. Bilateral fibro-caseous cavernous.

Since the end results of pneumothorax treatment are so intimately associated with the status of the contralateral lung, we classified its intergrity in the first six groups into five sub-groups as follows:

1. Essentially negative.
2. Deep peribronchial infiltration.
3. Disseminated bronchogenic extensions, caseous pneumonic and caseous bronchopneumonic infiltrations.
4. Active fibro-caseous infiltration.
5. It is apparent that the entire classification is a correlation of the clinical, physical and roentgenological findings.

Since the causes of failure are also dependent upon the degree of collapse we further classified our pathological groups according to the degree of compression as follows:

1. Satisfactory Compression.
2. Partial Compression.

3. No Free Space.

By "Satisfactory Compression" we mean that a sufficient degree of compression of diseased lung tissue was produced, which, according to our experience, if maintained, would render the compressed lung, as a source of any symptom, improbable. We refer particularly to such symptoms as persistence of germ laden sputum in quantities amounting to twenty-five or thirty cubic centimeters or more daily, fever of one hundred or more, and tuberculotoxemia over a period of two or three months after beginning pneumothorax.

It has been our custom after having established a satisfactory compression, to maintain it rather than to produce a total collapse, which is often productive of serious complications and responsible for many failures.

A total collapse of the lung is necessitated in some cases but there are many cases wherein a compression, thought satisfactory, does not involve a total collapse and may amount only to a partial collapse.

Under the term, "Partial Compression" we placed all cases where a satisfactory compression was unattainable, adhesions being our greatest obstacle. For instance, even though an almost total collapse of the lung was produced, if we were unable to satisfactorily compress diseased lung tissue or to close cavities because of adhesions, the case was placed in the "Partial Compression" group, for the obvious reason that the degree of compression was not satisfactory.

In connection with "No Free Space", no explanation is necessary except that cases have not been placed in this class until after repeated attempts over all lobes of the lung, proved the absence of free pleural space.

In grouping our cases in relation to Causes of Failure, we have classified them into two groups; those wherein there was improvement in many instances to the extent of clinical recovery but event-usually terminating fatally, and a second group wherein the disease was progressively fatal, in spite of treatment.

Most interest centers in an analysis of the Causes of Failure in the Improved but Fatal Cases, of which there were eighty-four

out of the six hundred comprising this study. The causes of failure in this group in the order of their frequency were as follows:

1. Adhesions preventing satisfactory compression....23 cases
2. Extraneous causes (not related to tuberculosis....12
3. Unsatisfactory social and economic conditions....10
4. Progression of disease in contralateral lung..... 9
5. Treatment abandoned voluntarily..... 8
6. Post-pneumothorax progression of disease in contralateral lung 6
7. Enteritis, laryngitis and other tuberculous complications not dependent upon treatment 5
8. Tube drainage of purulent exudate..... 3
9. Aspiration tuberculosis following hemorrhage.... 2
10. Widespread character of disease 2
11. Early obliterating pneumothorax 2
12 Spontaneous pneumothorax..... 1
13. Sudden death to post-pneumothorax hemorrhage.. 1

Of our six hundred cases, there were one hundred and eighty-six progressively fatal in which a pneumothorax was attempted or established. The causes of failure in the order of frequency in this group were as follows:

1. No free space ..... 72 cases
2. Widespread character of disease (therapeutic pneumothorax only-Ultimum Refugium cases..... 62
3. Adhesions preventing satisfactory compression.. 23
4. Serious tuberculous complications (enteritis, laryngitis, meningitis) 14
5. Progression of disease in contralateral lung..... 13
6. Spontaneous pneumothorax 2

The most frequent cause of failure in our Improved but Fatal Group (eighty-four cases) was our inability to establish a satisfactory Compression (23 cases) and in our Progressively Fatal Group (186 cases) it was our inability to find free pleural space (72 cases). In both groups death was due to the independent evolution of the tuberculous process.

Of our six hundred cases we established a satisfactory Compression in approximately forty percent. In approximately forty percent we established a Partial or Unsatisfactory Compression and in twenty percent, after repeated attempts, we encountered no free pleural space.

Of the six hundred cases, there was a total of two hundred and seventy Improved but Fatal and Progressively Fatal, or forty-five percent, and of this number in one hundred and eighteen cases, or forty percent, adhesions prevented a satisfactory compression, or no free pleural space was encountered. In the eighty-four Improved but Fatal Cases, there were twenty-three in which adhesions prevented satisfactory compression and in the one hundred and eighty-six Progressively Fatal Cases, there were also twenty-three cases in which adhesions prevented Satisfactory Compression but seventy-two cases in which no free pleural space was encountered.

According to our experience, practically no tuberculous lung, diseased to the extent of requiring a pneumothorax, is free from adhesions. They are not all demonstrable roentgenologically nor by physically diagnostic means. However, by means of the thoracoscope, we have demonstrated many adhesions which were not visible on x-ray plates. This is also a common post-mortem observation. Adhesions may be encountered and cause interruption at any phase of pneumothorax treatment and it is impossible by any method to always foreteli their presence. When they are encountered and prevent a Satisfactory Compression, treatment should be energetically carried on by very frequent inflations, caution being exercised to avoid too high pressures. By this means they are often separated or stretched, frequently permitting satisfactory compression and other times combatting an early obliterative pneumothorax by maintenance of a sufficient degree of pressure. Proper judgment should be exercised in using high pressures in dealing with adhesions, on account of the danger of too rapid separation, causing exudates or tearing the lung cortex and the consequent production of lung perforation, empyema, etc. Much more can be said regarding adhesions but the scope of this paper will not allow of even a brief discussion of all phases of the subject.

Extraneous causes was next in order in frequency in our Im-

proved but Fatal Cases and included such cases, as death from disease not related to tuberculosis, operative procedures, death due to accident, etc., 12 cases. Some of these patients were clinically well and it is important for us to include these as a cause of failure, since they have influenced in an unfavorable manner our pneumothorax end results, being responsible for fourteen percent of the deaths.

Unsatisfactory Social and Economic Conditions (ten cases, 11%) included such causes as limited finances, requiring patient to resume activities too early. Many of these were wage earners and housewives who were obliged to resume household duties, oftentimes living under very unfavorable circumstances during their ambulatory treatment.

Progression of disease in contralateral lung was responsible for twenty-two failures, (approximately 7%). Nine failures being among he eighty-four Improved but Fatal cases and thirteen failures among the one hundred and eighty-six Progressively Fatal cases.

Of four-hundred and eighty cases in which a partial or satisfactory compression was obtained two hundred (approximately 41%) had active or quiesent fibro-caseous infiltrations or disseminated bronchogenic caseous extensions in the contralateral lung demonstrable by physical or roentgenological methods and eighty-two cases (17%) had desolate bilateral or bilateral fibro-caseous cavernous type of disease, yet notwithstanding this, in only thirty-five instances (approximately 12%) did progression of disease take place in the contralateral lung of which twenty-two were fatal.

In view of the fact that two hundred and eighty-two (58%) out of four hundred and eighty in which a pneumothorax was established, had demonstrable disease in the contralateral lung, it is apparent that the percentage developing a progression of disease in the non-compressed side is surprisingly small (12%), but it is striking that when progression did take place the mortality was exceedingly high (60%).

The above figures may appear contradictory, as it would not seem reasonable to assume that out of two hundred and eighty-two cases of demonstrable disease in the opposite lung only thirty-five experienced a progression of this disease and out of the thirty-five cases, twenty-two terminated fatally. However, one must consider that

the limitations of diagnostic procedure are such that it is not always possible to detect the minor or questionable degrees of progression in an already diseased area.

Undoubtedly such minor progression took place in some of our cases early in their treatment but in the thirty-five cases of progression referred to, it was of a persistent, unquestionable and readily demonstrable nature of such a degree as to require discontinuance of pneumothorax.

We cannot too strongly emphasize the importance of careful and constant observation of the contralateral lung during the whole course of pneumothorax treatment, in order to detect the earliest manifestations of progression of disease or fresh invasion.

This can only be done by careful and searching examination, utilizing clinical, physical diagnostic and roentgenological procedures. From a clinical standpoint, in the presence of a satisfactory compression, any unexplainable fever or unexplainable quantities of bacilli-laden sputum should put one on the alert as to changes in the opposite lung.

From a physical diagnostic standpoint, auscultation will supply the most reliable evidence. Special attention must be given to the perihilar region and the hiliapical pathways, anteriorly and posteriorly and the upper auxiliary region.

Sharpened vesicular and broncho-vesicular breathing should be regarded with suspicion, although it must be differential from the compensatory type of breathing which is almost always present.

The presence of rales on expiratory cough, especially those associated with early infiltration we believe is best regarded as proof of invasion. Unless one records accurately the nature and topographical distribution of physical findings and compares these findings at each subsequent examination, extensive changes can take place before its discovery.

It should be the custom to examine the opposite lung before each inflation and, as often as it is deemed necessary between inflations, to detect any threatened extension or progression of disease.

By extensive employment of the x-ray, many cases of failure will be avoided; however, one must not only study roentgenograms

but also the fluoroscopic image, because each has its special advantages.

It has been our custom to have plates after every third or fifth inflation, and after the first month to make fluoroscopic examination before each inflation. Both methods of examination being carried out during the entire course of treatment.

Plates are of a particular value, inasmuch as they furnish a permanent record. One should not be content with the examination of the last plate made but it should be studied and compared with the preceding plates made on the case. The x-ray plate will supply evidence regarding the degree of compression, not only of diseased lung tissue but of healthy lung tissue; the presence of adhesions preventing satisfactory compression as well as their character, location and extent.

Adhesions are almost invariably present over superficial cavities, attaching the diseased lung to the chest wall in varying degree of fixation. If adhesions are dense, they render separation of diseased lung impossible and consequent failure to close cavities.

If adhesions are not too dense, separation of the diseased lung *en masse* may be effected. If adhesive bands are present, holding cavities in an expanded position by continuous inflations and constant roentgenological control, one is frequetly able to observe the stretching and separation of these adhesive bands, also is able to study the influence of certain pressures on the mediastinum and diaphragm which are necessary to the stretching and separation process.

If any portion of the compressed lung is held attached to the chest wall, even by adhesive bands, sound waves are conducted to and diffused over that region of the chest wall; and if one depends on auscultatory findings alone, one will be led to erroneous conclusions. Thus one will unknowingly produce dangerous displacement of the mediastinal contents, seriously jeopardizing not only the contralateral lung but the life of the patient and furthermore cause serious gastric disorders by extreme downward pressure on the diaphragm.

In avoiding lung perforations, another serious cause of failure, great care should be observed in the stretching and separation of adhesions, as the adhesion may represent the stretched wall of the

cavity, and its separation is not without danger of producing lung perforation. Separation of the lung from the chest wall is always beset with danger of tearing the diseased lung cortex and the subsequent production of lung perforation, bronchial fistula or empyema, which are also frequent causes of failure.

The separation of adhesions is nearly always attended with exudate but if the exudate remains serous it is never a cause of failure and even if purulent, is seldom a cause of failure, purulent exudate being a cause of failure in only five of our cases, three of which passed from observation and were submitted to open operation.

We firmly believe that adhesions are present in all cases demanding a pneumothorax, the first inflation frequently affecting a separation. They may not be demonstrable roentgenologically on account of their delicate nature but their presence can be proved by thoracoscopy. This view is also supported by autopsy statitistics. We furthermore believe that frequent careful roentgenological examination will reveal the presence of exudate in every case at some time during the period of pneumothorax treatment. The amount may be so small and so transitory that unless especially sought it will escape recognition.

In our series we have not included minimal transitory exudates, classifying only those wherein the quantity amounted to approximately fifty cubic centimeters or more, and this occurred in thirty percent of our cases.

Deep seated, centrally located disease, especially in the perihilar region, frequently escapes detection by physical diagnostic procedures but the x-ray will supply us with objective evidence of the most valuable character.

However, one must be able to differentiate those shadows produced by extension or progression of disease in the contralateral lung from those shadows due to condensation of lung tissue, the result of compression, and those due to circulatory changes, causing passive hyperemia.

It must be borne in mind that auscultatory findings are transmitted from the compressed lung to the opposite side of the chest, either by means of the bony frame work or through the condensed lung tissue itself, but the x-ray will prove the absence of any path-

ology, thus proving that we are dealing with transmitted phenomena.

Transmission of auscultatory findings from the compressed to the contralateral lung are more or less constant and if serious mistakes are to be avoided one must be able to differentiate auscultatory findings arising from the actual pathology over a given area from transmitted auscultatory phenomena.

The fluoroscopic control is very essential if one is to avoid grievous error, for if dependence is placed upon manometer pressure readings alone in regulating the amount of gas, one will be greatly misled, as every individual is a law unto himself as far as the reaction of his thoracic contents and absorbability of gas is concerned.

For example, two individuals of equal lung volume are given 250 cc of gas, with similar manometer readings, at three-day intervals and after the third inflation a plate is taken which may show that "A" has almost total collapse of the lung while "B's" lung is compressed but a few inches from the chest wall; and again, one may be surprised to find, after a few inflations, with uniformly low pressures that great displacement of mediastinum and downward pressure on the diaphragm has been produced.

One obvious advantage of the fluoroscope is that it permits visualization of all planes of the lungs by rotation of the patient under the screen, enabling one to visualize the pneumothorax cavity. Permanent records of the fluoroscopic examination can be made easily by making orthodiagrams which can be filed with the case record and referred at each subsequent inflation.

Treatment abandoned voluntarily was responsible for eight of our failures out of eighty-four cases, in the Markedly Improved but Fatal Group. Under this heading we have included cases wherein patients discharged themselves voluntarily from treatment believing themselves cured. This also includes patients who, because of their apparent health received misguided advice from relatives and friends who prevailed upon them that no further treatment was necessary.

Progression of disease in the contralateral lung; post pneumothorax, occurred in six cases long after the termination of a successful pneumothorax on the diseased side. All terminated fatally. This observation is interesting in view of the work of various observers who have demonstrated that a pneumothorax in the presence of a

flexible mediastinum the intra-pleural pressure is equal on both sides, thus explaining the beneficial influence which frequently takes place in the contralateral lung from compression of the worse diseased side. This is in accordance with our experience. It is not unreasonable to suppose that the abolishment of the beneficial influence of an artificial pneumothorax may have been a factor in the exacerbation of disease already present in the contralateral lung.

It is further worthy of observation that out of four hundred and eighty cases wherein a Satisfactory or Partial Compression was induced two hundred and eighty-two cases had unquestionable disease in the contralateral lung, yet in only thirty-five instances did the disease progress. This has an important bearing on the selection of material for artificial pneumothorax, since it has been held by many observers that the presence of well-defined disease in the contralateral lung contra-indicates pneumothorax treatment. It is possible that this attitude is too conservative but it must be emphasized that success is frequently proportionate to the degree of watchfulness devoted to the contralateral lung as well as judgment regarding the proper degree of compression.

Enteritis, laryngitis and other complications not related to pneumothorax treatment were the cause of failure in five case out of eight-four Improved but Fatal Cases and fourteen cases out of one hundred and eight-six Progressively Fatal Cases.

These figures are small when it is considered that out of four hundred and eighty compressed cases eighty-nine presented a well defined tuberculous laryngitis. Although we have classified both our laryngitis and enteritis cases together, as a matter of fact tuberculous laryngitis was a cause of failure in only one case, the remaining four having been caused by tuberculous enteritis and other complications such as meningitis, etc. Forty-four of our compressed group presented a well defined tuberculous enteritis. Although we realize that autopsy shows practically all cases of pulmonary tuberculosis which has existed any length of time have a tuberculous infiltration of the lymph follicles of the intestines, only those cases have been considered as tuberculosis enteritis which presented the well-known symptom complex and supported by the physical and roentgenological findings which are associated with this condition. From a

clinical standpoint it is doubtful whether any patient should be subjected to an artificial pneumothorax who has an enteritis so severe as to interfere with proper nutrition, especially if the enteritis does not respond to treatment.

We feel from our experience that tuberculous laryngitis, unless so severe that the patient cannot be nourished, is not a contra-indication for pneumothorax.

Tuberculous enteritis when well defined must be considered as a much more serious complication and will be a frequent cause of failure, particularly in those associated with ulceration and diarrheal movements. However, those cases of the chronic hyperplastic type offer a much more favorable outlook.

Twenty-nine of our cases have other serious tuberculous complications which included several cases of urogenital tuberculosis, yet it was a cause of failure in only one case.

Sixty-three out of four hundred and eighty compressed cases developed a purulent exudate. As a result of our observations during the past few years, we believe that nearly all purulent exudates occurring during pneumothorax treatment are tuberculous and this can be demonstrated in a great many instances by animal innoculation. These cases were treated for the most part by aspiration, or aspiration and irrigation combined, and a few by means of air-tight tube drainage; in none of these was the purulent exudate in itself the cause of failure. However, three cases developed a purulent exudate while away from our immediate observation and were operated by the open method by surgeons, death later ensuing. The open operation for a tuberculous purulent exudate is to be strongly condemned. If tubes are inserted, air-tight drainage is imperative.

Eighty-two cases (30%) out of four hundred and eighty compressed cases developed serous exudate in amounts upward of 50 cc; however, it was not a cause of failure in any instance.

Aspiration tuberculosis following hemorrhage was a cause of failure in two cases in the Improved but Fatal Group. Hemoptysis was a complication in seventy-nine of our cases before treatment; severe hemoptysis occurred in ten cases during treatment, eight of whom had only a partial compression; aspiration tuberculosis developed following hemoptysis in four of these cases, two of whom died.

Widespread character of disease was the cause of failure in only two of our Improved but Fatal cases. In the Progressively Fatal Group we attributed failure to widespread character of disease in sixty-two instances out of one hundred and eighty-six cases. Of these sixty-two cases of failure, forty-four belong to the desolate bilateral or *ultimum refugium* group; six had bilateral fibro-caseous cavernous tuberculosis and seven were of the severe acute infiltrating type of disease. Therefore, in all the other types of tuberculosis with which we have had to deal, we have attributed our failure to wide spread character of disease in only five instances.

Early Obliterative pneumothorax was a cause of failure in only two cases. It has been our policy when possible to maintain a pneumothorax until Obliterative Pneumothorax ensues but in some instances, particularly those complicated by adhesions and secondary pleuritic thickening, re-expansion occurs early.

While Obliterative Pneumothorax takes place frequently, it is evident that even short periods of compression are beneficial because we have had only two failures due to this cause.

Lung Perforation was a cause of failure in three cases. One in the Improved but Fatal Group and two in the Progressively Fatal Group. Lung Perforation during the course of artificial pneumothorax occurs with much greater frequency than is generally realized, for the reason that some are of slight severity, being almost devoid of symptoms and recognized only by careful study and observation. The symptoms are largely dependent upon the presence or absence of adhesions the size and character of the opening, also whether of the valve type or not. In our series of cases we recognized sixteen instances of unquestionable lung perforation as a complication of an artificial pneumothorax.

Sudden death due to post pneumothorax hemorrhage was the cause of failure in one case, the source of hemorrhage being unknown. This case had been under artificial pneumothorax for one year and had remained clinically well for another year following discontinuance of the pneumothorax when a sudden severe hemophysis occurred, proving fatal.

No Free Pleural Space was a cause of failure in seventy-two

instances out of four hundred and eighty Progressively Fatal Cases and was the most frequent cause of failure in this group.

Summary and Conclusions.

Adhesions are the most frequent cause of failure of artificial pneumothorax being responsible for forty per cent of failures. They are present in almost direct proportion to the duration and intensity of the disease causing over twenty-seven per cent of the failures in earlier cases which were Improved but Fatal and over fifty per cent of the failures in the more advanced cases which were Progressively Fatal.

Artificial pneumothorax exerts a beneficial influence upon active disease in the non-compressed lung if it is not too extensive. While fifty-eight per cent of our cases had active or quiesent disease in the non-compressed lung only twelve per cent were unfavorably influenced.

Progression of disease in the non-compressed lung was responsible for approximately seven per cent of our failures.

Serous exudates occurred in practically every case under pneumothorax treatment but as long as it remained serous it was never a cause of failure and even when becoming purulent it was seldom a cause of failure.

Tuberculosis enteritis when so severe as to interfere with the patient's nutrition and unresponsive to treatment is probably a contra-indication for artificial pneumothorax.

Tuberculosis laryngitis unless so severe that the patient cannot be nourished is not a contra-indication.

Lung perforation and obliterative pneumothorax were infrequent causes of failure.

Success in the treatment of pulmonary tuberculosis by means of Artificial Pneumorthorax is largely dependent upon its earlier application.

Cases of a character clinically giving unsatisfactory results by the usuel sanatorium routine should have the advantage of an artificial pneumothorax early at a time when adhesions are less likely to prove a cause of failure.

DISCUSSION

Dr. Packard: Pneumothorax may spread in the so-called good side, but so frequently the good side spreads in the treatment. The disease progresses whether tuberculin is given or not. I would like to ask Dr. Matson as to the effect of pneumothorax to control hemorrhages. In my own experience it has not been favorable. The danger has already been done by the time the pneumothorax is performed.

Dr. Wilson: I had one experience in not limiting the amount. One of the internes found the apparatus was working so well that he kept on going. I was called and he had given about 1000 c. c.

Dr. Miller: It seems to me perhaps that we are allowing too many cases to fail. Another possibility in order to obtain a collapsed lung is the cutting of the adhesions. We should give the patient the opportunity of this operation. We have come gradually to use artificial pneumothorox more experimentally than anything else, and we are finding a considerable percentage that arrests the activity of the disease. Reviewing a group of cases to find how many were doing well, we found some of them working who within a year before were certainly doomed to death within a few months. Although they will eventually die of tuberculosis, we have given them a new lease of life.

Dr. Webb: What type of cases are these that Dr. Matson speaks of? How long is it kept up? With the people who have some means to combat this disease we are in favor of postural rest, rather than pneumothorax. We have to decide what we can give according to the patients. Some of the doctors have kept on taking it, and the reason for this is because they got it free.

Dr. Rugers Remarks made by Dr. Miller bring me to my feet to discuss this most interesting paper. Like Dr. Miller, we have had some success in compression of the lung for a tuberculous pneumonia. On paper it has always seemed that this would be rather out of the question, as it seemed quite impossible to compress a solid lung just as it would be impossible for one to compress fluid. Nevertheless it seems to work to a certain extent and I have seen more than one case where a great deal of good is accomplished by the use of pneumothorax in the conditions.

Our results with hemorrhage have been both satisfactory and unsatisfactory. In some cases it has unquestionably saved life. In other cases, it has despite the fact of our having obtained a definite compression, aggravated the condition and made the bleeding worse. These cases were I might add cases in which there was no question as to where the bleeding was coming from. The non-bleeding side could not possibly have been compressed instead of the bleeding side. It is a question in my mind whether we should not be more ready to compress the lung for hemorrhage. As a rule we wait until everything else has failed and we have an exhausted patient to deal with. It is not fair to the patient and it is not fair to the procedure. Were we, as a temporary measure, to compress the lung more promptly after an hemoptysis, keeping it

firmly compressed only sufficiently long to assure us that we have a firmly established clot and then letting it re-expand, I think our statistics on beneficial results for a control of hemorrhage by pneumothorax would appear considerably better.

Dr. Lichty: Regarding pneumothorax, in what condition are those patients? We hear of various complications, but what is the ability of these patients to respond to this treatment? Is it not important in considering this procedure to give some clear picture of the state of the patient on whom it is being done?

Dr. Matson closing disucussion: Limited time prevents my bringing out many points I should like to discuss in detail at this time.

Our pneumothorax material is large because Portland is the medical center of the Far North West, thus drawing from a territory twice as large as New England.

All pneumothorax cases were patients under treatment at the Portland Open Air Sanatorium and the Tuberculosis Clinic of the Medical Department of the University of Oregon. Both services are very large by virtue of having been the first Tuberculosis Sanatorium and Free Tuberculosis Clinic established in the Pacific Northwest.

We have treated no early cases of tuberculosis by Artificial Pneumothorax. Of the six hundred (600) cases reported by us eighty-five (85) were second stage N. T. A. and five hundred and fifteen (515) were third stage N. T. A. cases.

The best results are obtained in the earlier types of cases where satisfactory compression can be obtained, and the less the disease in the contralateral lung, and the less its activity the better the prognosis. The question of prognosis in various types of disease will be presented in two (2) papers at the forthcoming meeting of the N. T. A. and the A. M. A. next month. However, I may briefly state here regarding the end results in various types of tuberculosis that among the six hundred (600) cases there were one hundred and ninety four (194) chronic Fibro-caseous types with little or no excavation but progressive in character, (one hundred and eighty-nine had tubercle bacilli in the sputum) of which thirty-five (35%) percent are now clinically well, and thirty-five percent (35%) dead; others of course are arrested in ambulant, favorably influenced, temporary improved or unimproved. While of one hundred and seventy seven (177) chronic Fibro-caseous Cavernous progressive types of tuberculosis twenty-two and one half (22½%) are clinically well and thirty-six percent (36%) dead and of thirty-nine (39) severe acute infiltrating types, caseous pneumonias, and caseous broncho-pneumonias, eighteen percent (18%) are well and sixty percent (60%) are dead. While of eighty-four (84) extensive bilateral cases all are dead.

However, aside from the type of case, the degree of compression, and the status of the contralateral lung are factors of tremendous influence for good or bad. For instance, the outlook, in a chronic fibro-caseous cavernous

case with satisfactory compression, and essential freedom of infection in the contralateral lung, is better than the outlook in a chronic fibro-caseous case with little or no excavation and essentially negative contralateral lung, but with only partial compression. Again the first type of case has a better outlook than the latter type, with a satisfactory compression in the presence of disseminated bronchogenic caseous extension in the contralateral lung.

Of the six hundred (600) cases ninety-seven (97) had hemoptysis before the artificial pneumothorax was instituted, while only ten (10) had hemoptysis afterwards.

We use the Forlanini Saugman technic (modified) utilizing a Solomon pneumothorax catheter of very small calibre, introducing it into the pleural cavity through a small cannula with a flat top, like a push tack. This instrument is a little larger than the usual pneumothorax needle. On the other hand, in spite of Dr. Johnson's remarks, we regard it as the safest instrument to use. One knows at once, the small push tack-like cannula is pushed through the chest wall whether adhesions are present or not. Furthermore we have our Solomon catheter nibs made to fit a Leur syringe, so that when adhesions are encountered we may attempt forcible separation by means of salt solution injected with 50 c. c. syringe through the catheter. In case adhesions cannot be separated we frequently introduce hypertonic salt solution, five to ten percent into the pockets between the visceral and parietal pleura. The hypertonic salt solution sets up an inflamatory process resulting in further pleuritic thickening followed by secondary retraction which serves to splint up the affected side and rest it.

Regarding dosage an interval of gas, we introduce small quantities one hundred fifty to two hundred (150 to 200) cc at five (5) day intervals and after three to five injections we plate steroscopically routinely every case, after which the interval and quantity is changed according to the case.

We routinely fluoroscope every patient before compression and make an orthodiagram to check each inflation against subsequent ones.

THE ORGANIZATION OF CLINICS AND CLASSES IN CHAUTAUQUA COUNTY

By WALTER L. RATHBUN, M. D.

At the 1922 fall meeting the Board of Supervisors of Chautauqua County granted permission to the Board of Managers of the Newton Memorial Hospital to employ a county tuberculosis nurse. A tuberculosis nurse was on duty until December 31, 1914, when the position was abolished. Owing to unavoidable complications in connection with the appointment of the one we had selected for the position, our county nurse did not report until April 1, 1923. This delayed us greatly in starting in on our program. However, the Chautauqua County Tuberculosis Committee with its usual fine spirit, voted to have its Executive Secretary, a nurse, cooperate with the county nurse in the organization of clinics and classes so that much time has been saved.

Chautauqua County has a population of about 115,000 and over 60,000 of this number reside in the cities of Jamestown and Dunkirk where the tuberculosis work is well covered by efficient Health departments. The remainder, nearly 55,000, live in villages and distinctly rural sections covering an area 36 miles long by an average of 30 miles wide, where clinics have been held from time to time each year by the Executive Secretary of the County Tuberculosis Committee. This section was selected as the most promising field for the work of the county nurse. The clinics held by the County Committee were well distributed and the attendance was satisfactory, but it was not possible to cover the territory properly with the force at hand.

In laying out the work of the county nurse we decided to make repeated canvasses of the county and to facilitate her operations, as well as to render equal service to all sections, thereby eliminating any possible criticism, the territory was divided into ten zones with from one to three centers of operation or headquarters in each, the number of centers depending upon the density of the population.

The tuberculosis hospital which is the base of operations is located northwest of the center of the county.

This scheme was discussed with a large number of the representative medical men of the county before it was put into operation and their support was promised. I can only add that their promise has been kept very faithfully thus far. The nurse visits the centers and the surrounding territory on two successive days before the clinics and classes are held. She calls on the known cases, including, of course, the ex-patients and their families. The doctors, clergymen, school teachers, lodge leaders and the older residents are interviewed and are requested to furnish information about possible cases. An effort is always made to get into touch with those who have been exposed to massive doses of tubercle bacilli and to arrange for their attendance at the clinics. Later, we intend to appeal to such cases through the local papers with the hope that some unknowns can be induced to come for at least one examination. There is at times a slight overlapping of territory, as might be expected, but this has proved to be of advantage rather than otherwise. Clinics and classes are to be held in each one of these centers every two months with the hope that eventually we will come into intimate contact with all who are in need of chest examination

We found that some who were in need of a chest examination were afraid to come owing to the fear that we might find traces of tuberculosis. There also seems to be a fairly widespread impression that medical men who are especially interested in tuberculosis will make a diagnosis of tuberculosis on most of those who come to them for an opinion. This feeling is so marked in one place that only recently our county tuberculosis nurse was informed that those who hold clinics are a "bunch of fakirs." It is hoped that, owing to our periodical visits to the various centers throughout the county, people in the villages will become familiar with exactly what we are trying to do and will come to clinics as they do in the cities. I believe it is simply a question of living down the erroneous ideas that have gained a foothold in some sections.

One advantage resulting from holding clinics in sequence in different centers throughout the zones lies in the fact that some prospective patients are diffident about coming to a clinic in their own

center but will journey to an adjacent one so that they can be examined without the curious eyes of the village gossips following them to the clinic door. When clinics are held at convenient distances such cases are reached and, in addition, those who through previous engagements are not able to come to their home clinics may be able to attend one at another nearby center. After the routine becomes well established the people will know when to expect us in their zone; also the territory we will be working on any particular day. A repetition of clinics in the various zones will enable us to keep close watch of the known cases, particularly ex-patients. We are trying to follow out Dr. Pratt's class idea, modified to fit our needs, and are organizing classes which, of course, at the present time are small. The number varies from one to four in the centers with a total thus far of sixteen. However, these are bound to increase as our patients are discharged and such classes will furnish an ideal way of keeping in touch with our ex-patients. Arrangements are being made to hold classes in Jamestown and Dunkirk during the evening so that those who are working can be present. Many of these cases are being examined periodically at the clinic and take up a great deal of time that might be used for examining new cases. More could be done for them if a special class were formed and monthly or bi-monthly sessions held. Eventually we expect to have a total of from forty to fifty class members in these two cities.

A small pamphlet has been prepared for the members of the class and this contains most of the essentials for use in home treatment. I think that we can depend upon our class members to popularize the movement as they appear to be anxious to cooperate. While working the various centers we visit those patients who are confined to their beds. Before starting the classes I went over the matter with various physicians throughout the county to find out if in their opinion there would be any criticism on the part of the profession. I had feared that possibly the medical men might feel that we were interfering with their practices. However, the universal opinion was that the doctors would be only too glad to cooperate and in most instances would be in favor of our taking over the entire treatment of the case. As one physician expressed it: "A general practitioner never makes a reputation in treating cases of tubercu-

losis and any of them will be glad to get rid of the job." However, under the present organization it is obviously impossible for the man who is running the clinics to take entire charge of the case although, of course, we are glad to assume a good share of the responsibility. It is believed that class members will be of great service in referring patients to the clinics for examination. It will not be many years before our out-patient service will equal, if not exceed, the work that is being carried on at the hospital for if our plans work out, we will have what will be practically a county-wide tuberculosis institution on the cottage plan. The members of the classes will be examined, advised and at each examination the importance of the continuance of treatment will be reiterated. Many patients forget quite promptly what they learned during treatment at an institution and even when they do not forget, there comes a time when their relatives refuse to believe that they are not well. When there are signs of exacerbation patients will be admitted to the hospital. We have a plan in mind of setting apart wards for each sex for intensive rest treatment and heliotherapy over short periods for class members. A tent colony, to be located near the hospital, for the use of class members during the summer vacation period, has been considered and may be worked out later. Such a colony would do much in popularizing the class movement.

We have found that there is wide-spread phthisiophobia in the county and we are bending our efforts towards disseminating the truth wherever we go to combat it. I believe that our frequent meetings with men and women of influence in the community together with our dealings with the rank and file will eventually do much towards eliminating this opinion. Recently one of our new patients begged us not to make known her condition as she would be ostracized by the entire community. She stated that her father died of tuberculosis and while he was sick her home was not visited even by the neighbors with whom she was intimate. Occasional clinics held at random throughout the county would have little effect in dissipating any such idea for more than a temporary period, whereas it would seem as if a continual round of visits at periodic intervals would finally do the trick. The plan outlined practically doubles the work and responsibility of the hospital superintendent and if

the work grows it may be necessary to have additional help from time to time. The State Department of Health can be relied upon to furnish assistance whenever possible and the State Charities Aid Association have been most cooperative in connection with clinic work, so that I believe that insofar as Chautauqua County is concerned the superintendent of the tuberculosis hospital can handle the large bulk of the work. The co-ordination of the hospital and outpatient service is much facilitated when one man is in control of both and the hospital beds can be utilized to the best possible advantage. After we become familiar with the ground and the people in general, it is planned to trace out and examine the members of all families where a death from tuberculosis has occurred. Dr. McSorley of the N. Y. State Health Department has already furnished me with a list of deaths from 1915 to date. This can be done rather easily in a county like Chautauqua where, outside of the cities, the population movement is fairly well stabilized. In the hilly rural sections we have found a relatively large number of cases of the fibroid type. One new case discovered recently has had symptoms of tuberculosis for over fifty years. She has left no discoverable trail as all of her family, including her husband, are dead. Her children died of acute infectious disease while young. However, the fresh trail of some of the chronics can be picked up in various locations throughout the county and I believe some good hunting and interesting experiences lie ahead of us.

The difference in the type of disease between the rural sections and the cities in the county is most marked. The majority of the new cases found in the cities are of the acute or active type, whereas we have not found one new case outside of the cities where there have been clinical signs of activity. Only recently I examined a boy in a country village situated in the hills who had the disease for from five to six years. He was 16 years old, very much undersized and had involvement of the entire right lung with signs of cavitation in the right upper lobe. His chest showed all of the earmarks of an old chronic fibroid. The location of the cities, however, has nothing to do with the type of infection, as Jamestown is located in a hilly section with a good altitude. I am assisting Dr. J. J. Mahoney, Commissioner of Health of Jamestown, in his tuber-

culosis clinic work and at the last clinic I attended we found three new cases of tuberculosis, all of them advanced and running a temperature.

Judging from our experience to date I believe that much more can and should be done for ex-patients and for the tuberculous who will not accept treatment in a sanatorium or hospital. After all, in spite of its great value, which cannot be over-estimated, sanatorium or hospital treatment leaves much to be desired after the return of patients to their homes. Frequently the lessons learned there are soon forgotten until symptoms of a relapse appear and then many times it is too late for further successful treatment. In addition, occasionally the ex-patient has to be protected from interested relatives and friends who are strong on advice but weak on knowledge concerning tuberculosis. We feel that our periodical meetings with the ex-patients will help them in the solution of their various troubles and we hope that in some cases we may be instrumental in favorably influencing the course of the disease. Every relapse that is staved off and every patient that can be given home treatment means a saving to the tax payers of the community, while the expense of carrying out a county-wide supervisory out-patient service on the modest scale that we have attempted will be relatively small and should not be objected to by the Board of Supervisors of any county.

On our next visit to some of the zones we plan to take several patients with us and before the clinic is held these will be demonstrated to any of the local physicians who can arrange to come. x-ray films of these cases will be shown after each examination. In addition to the possible educational value it is hoped that such demonstrations will stimulate and keep alive the interest of the physicians in our clinics. These demonstrations will be repeated from time to time if the satisfactory results are obtained.

DISCUSSION.

DR. MILLER: I think that New York State has taken perhaps the lead in the development of work in county lines. The conception that the hospial is the center to work on tuberculosis is going to be a step of very great value. The Millbank Fund has started work in Cattaraugus County for the next five years at least. They have selected a county site, Syracuse, and probably will select one part of New York City. I think that we will see if these demonstrations are successful a considerable amount of public and private money spent in the development of better control of communicable disease.

SOME LESSONS LEARNED FROM COMPARISON OF CHEST ROENTGENOGRAMS AND PHYSICAL FINDINGS IN TWELVE HUNDRED CASES COVERING FOUR YEARS' EXPERIENCE WITH TUBERCULOUS EX-SERVICE MEN

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Following the entrance of Canada into the Great War, and the development of pulmonary tuberculosis amongst her troops, came the urgent demand for institutional treatment for those who were in need of care. As the Federal authorities had no sanatoria of their own, the various Provincial institutions throughout our country were asked to set aside as many beds as they could possibly spare for men who had broken down during service. The Nova Scotia Sanatorium was among the first to take in tuberculosis soldiers, and from a small unit of 32 beds it rapidly grew in size through the years of the war, to an institution capable of looking after 400 patients. During the past two years the number of ex-service patients has gradually fallen off so that we have reduced our accommodation to 290 beds including civilian and military.

Since the latter part of 1914, when the first ex-service man was admitted to the Nova Scotia Sanatorium, 1328 men have been admitted for treatment there, of whom 367 have been re-admitted, many of them several times. In addition, the Pension Commissioners have been sending their cases to us for observation and diagnosis, so that pension awards to these men can be determined from our findings. It may be added that a large proportion of these pensioners have been, at some time, patients of the Nova Scotia Sanatorium, and as they are regularly brought up every six months for re-examination, they are referred to us for advice, and their pension is graduated or treatment is prescribed according to our findings. This you see has given us an unexcelled opportunity to study respiratory affections in ex-service men and to observe the course of the disease

through physical examinations and serial x-ray plates over a period of years. It has also been the means of providing us with material for a close study of the causes bringing about relapse in 300 patients who have come back to the sanatorium for observation and treatment.

During the past five years the standard of our examination has been pretty much the same as that of similar institutions in America. and has included the following: (1) A full history of the patient's illness, especially as regards the causes of his breakdown in health and the relation of this to military service. (2) Complete physical examination. (3) Nose, throat and dental examinations. (It may be mentioned that all focal affections when present in the upper respiratory tract, are corrected and treated by specialists). (4) Laboratory examinations, including sputum analysis, complement fixation test and Wassermann test. (5) X-ray examinations, fluoroscopic and stereoscopic. (6) The gathering of all data obtained during the patient's period of observation, and correlating the clinical, laboratory and roentgenological examinations so that a diagnosis may be arrived at.

Of the 1200 cases included in the present study, the pulmonary condition on admission was as follows: Not tuberculous 332 or 27.7%. Suspected tuberculosis 42 or 3.5%. Positive tuberculosis 826 or 68.8%, including Minimal 267 or 32.3%. Moderately advanced 347 or 42% and Far advanced 212 or 25.7%.

Those of us who have had oportunity to examine and re-examine ex-service men since the year 1915 will recall the pre-conceived notions we had gathered from our training and experience regarding the value of history and auscultation findings in making a diagnosis of pulmonary tuberculosis, and we must confess that some of the signs upon which we had come to place so much dependence have not stood the test of experience with ex-service men.

What I mean is this. We have found that history and auscultation findings which in the past would have led us unhesitatingly to a diagnosis of definite pulmonary tuberculosis, now, in many cases, lead us to nothing more than a suspicion of definite disease, and many of these cases we finally have to classify as not tuberculous. The realization of this has been brought about by refinements in

diagnosis, by the free and careful use of stereoscopic chest roentgenograms, by more careful laboratory analysis of the sputum and of the blood, and by the unusual opportunity afforded for comparing these findings with our history and physical examinations checked up over a period of years.

With regard to history we have this to note. Excluding the cardinal symptoms, hemoptysis and pleurisy with effusion, to which I will refer later on, we find that 819 or 68.2% of the 1200 patients included in this study gave what formerly we would have considered a positive or a very suggestive history, that is several such symptoms as cough, expectoration, pains in the chest, loss of weight, strength and appetite, shortness of breath, sweats; but in only 551 or 67.2% of these could any tuberculous disease be discovered. Even a history of hemoptysis, which we are accustomed to trust as one of the deciding factors in diagnosis, frequently fails us in this type of case. Of the 1200 patients included in the present study 238 or 19.8% gave a history of hemoptysis (not including streaked sputum to which as a history finding we pay but little attention). Of the 238, 184 or 77.3% were found to have pulmonary tuberculosis, and 54 or 22.7% were not tuberculous. The X-Ray showed definite parenchymatous changes in 168 or 70% of those who gave a history of hemoptysis. This differs rather widely from the findings of Brown and Heise published in the report of the National Association for 1922 in which they state that 90% of their cases having a history of hemoptysis showed a definite parenchymatous lesion in the roentgenogram.

Knowing that the statistics of Brown and Heise are founded upon a study of 1000 civilian patients and that our figures are gleaned from a study of over a thousand ex-service men, we cannot but wonder whether or not the nature of the material studied is the ground for this notable difference in findings.

Our experience in this larger study is emphasized in a special "Study of Readmission and Relapse" which we have made, and we cannot escape the observation that history and symptoms in ex-service patients have different value from that of the same in civilians. That these symptoms are genuine in many cases, and honestly over-estimated in many others, there is no doubt. But where they are genuine, they are often out of all proportion to the amount of

discoverable disease, and they often accompany bronchitis or other respiratory trouble not tuberculous. This makes the ex-service type of cases misleading and difficult to deal with, and necessitates sending these patients to a sanatorium where special training and every facility can be brought to the examination, physical, clinical, laboratory and x-ray, and where some time can be spent. We keep these men at least ten days, and in dubious cases a month or more, for observation, to decide whether or not treatment is necessary.

The most dependable symptom is shown to be pleurisy with effusion. This was given in the history of 78, or 6.5% of the cases in our present study, and, of the 78, 71 or 91% were proven to have definite pulmonary tuberculosis.

So far as the usual symptoms are concerned, that is those of the group already referred to as occurring so frequently in the ex-service type of case, we are inclined to set a little less diagnostic value upon them, even in civilians, than we did in former years.

So much for the modification of our views regarding the value of history. Let me go on to mention a lesson we have learned regarding the valuation of certain physical findings, particularly slight change in the percussion note in the upper third of the lungs, which used to mean much more to us than it does today as an indication of the presence of tuberculosis. In fact, checking up with our x-ray plates has taught us that slight dulness even in the apices does not necessarily indicate the presence of tuberculous disease, or even of any respiratory disease. Definite dulness in this area, however, we still find usually accompanies some pathologic change in the lung or pleura. Further in auscultation I have been forced to modify my views regarding the value of the changes in breath sounds heard in the apices of the lungs, which formerly, to my mind, strongly suggested the presence of tuberculous infiltration in that area, but which do not now carry much weight in that respect, as we find they frequently accompany respiratory disease other than tuberculosis.

This sounds as if I believed in nothing but rales as an auscultation finding of definite value; and perhaps we are coming to the point where we must consider that apical rales are the only surely dependable auscultatory sign of pulmonary tuberculsis; but we

should not wait for rales to be heard before making a diagnosis of tuberculosis, because in an appreciable number of cases there is definite disease without the presence of rales, and this may extend over a large area before the rales are heard. On the other hand one must not be to ready to make a diagnosis of definite disease from a suggestive history and a few percussion and auscultation changes without the presence of rales and without positive x-ray findings. Indeed, in the type of case in which there are no rales nor positive laboratory findings, stereoscopic chest roentgenograms are a vital necessity to a correct diagnosis.

Having referred to laboratory findings, I may say that here also we have learned one or two lessons. To put it briefly, we see the need of much greater care in the microscopic examination of sputum. By care I mean, in apparently negative tests, not only the examination of specimens taken on several consecutive days, but the spending of a much longer time in the study of each smear. We agree with Pottenger that the three minute standard set by the National Association as the time to be devoted to a single smear is not sufficient for a correct report as to whether bacilli are present or absent. We have found that a five to fifteen minutes' search sometimes discovers bacilli in a specimen apparently negative in a three minute study. It may be interesting to note that, in a special study carried out in our laboratory, of two hundred and sixty-eight sputa apparently negative by the direct smear method of examination, it was discovered that, in every instance where bacilli could be found by the concentration test, they could also be found by the direct method, provided sufficient time was spent in the search. The conclusion seems to be that the accuracy of the direct smear method depends upon the length of time spent upon it. We have also decided from this study of ex-service men that it is advisable in all cases to give the Wassermann test. Out of 243 receiving this test 13 or 5.3% gave a positive reaction. In a considerable number of these, luetic infection appeared to be responsible for symptoms which might have been attributed but which were not attributable to tuberculosis. The auto-urine reaction of Wildholz we believe from a study of forty-six test cases, to have no value as a factor in diagnosis.

It is not my intention to discuss in this paper those x-ray shadows the meaning of which are fairly well accepted by roentgenologists, but there are a few points I would like to take up. In passing we may say that, in practically every case in which our clinical methods had demonstrated the presence of tuberculous disease, x-ray findings were in agreement. Two of the points upon which clinicians and roentgenologists are not entirely in agreement are the diagnostic values first of peribronchial thickenings especially when seen in the upper third of the lungs and second of minute nodal parenchymatous changes chiefly at the summit of the lungs. Similarly in our experience among the cases that have given us difficulty in reaching a diagnosis were found many of those in which the clinical findings were uncertain or altogether negative and the x-ray shadows in some showed thickened or infiltrated paravertebral trunks leading to the apices, in others minute nodal parenchymatous changes at the summit of the lung. The symptoms complained of were the same as those given by patients who have been proven to have pulmonary tuberculosis, and as a number of these men had before coming to us been diagnosed as either having suspected or actual tuberculosis and had been treated in various hospitals in Canada and England, this made the problem of diagnosis harder to solve. Some of these patients were finally diagnosed by us as having tuberculosis, others were suspected of having it, and others again were definitely established as not tuberculous. Later studies, clinical and roentgenological, extending over a number of years, showed us that a good many of these cases had actually been not tuberculous from the very beginning. We have come to believe as far as peribronchial thickening is concerned that, unless the increased accentuation and thickening of the trunks is accompanied by definite beading or by collateral haziness which may be interpreted as hyperaemia, these shadows seen in x-ray plates are of little significance. Certainly not every x-ray picture showing thickened or peribronchial trunks or minute opacities at the summit of an apex indicates clinical disease requiring treatment.

There have been difficulties, too, in interpreting correctly x-ray shadows seen in the hilus region. We agree with roentgenologists that increased fibrotic thickening of the hilus shadows, accompanied

by calcified nodes is of little clinical importance. However, when there is marked evidence of old infection such as large calcareous nodules, with more than usual evidence of old focal infection in the surrounding parenchyma, prolonged observation is necessary before clinical disease can be ruled out. In this connection we would like to note that only two cases in our present study have shown us markedly enlarged caseous hilar glands the picture of which could be called one of true hilus tuberculosis. Further, we have specially observed in twenty-five cases of the present series a small parenchymatous lesion in the 4th and 5th intercostal spaces, 17 in the left lung and 8 in the right, in every case the apex being clear of disease, and only twelve of which had positive clinical findings. These lesions were just outside the limits of the hilum and were evidently reactiviation of an old focus of tuberculous infection. I mention this type of case because this is an unusual location for tuberculous disease, and because, in the absence of positive sputum, the clinical evidence is insufficient and diagnosis is difficult, depending almost entirely upon x-ray findings.

In 163 cases diagnosed as bronchitis, apart from bronchiectasis which in many instances gives a characteristic x-ray picture, we have not established a definite radiologic appearance of this condition. Bronchitic and asthmatic cases of long standing can be readily recognized from the roentgenograms, the picture being one of enlargement of the hilus shadow, marked thickening of the descending trunks and increased linear markings, and of the peculiar wiry appearance so often associated with this type of case. This, with the depressed diaphragm and in some cases the increased ray penetration due to emphysema, generally serves as a basis for x-ray diagnosis; but in our 1200 cases increased linear markings were the rule rather than the exception, and we have yet to establish a definite x-ray picture on which we can base a diagnosis of bronchitis.

In 29 cases giving a history of gas poisoning we have been disappointed in the meagre x-ray findings obtainable. In severe gassing, a definite increase of lung markings has been noted; but in those giving a history of slight gas exposure, the x-ray picture has been the same as that of a normal chest or at the most of a bronchitic condition.

Tuberculosis as a result of gas poisoning we have found to be very rare, occurring in only one of these 29 cases.

Closely associated with bronchitis is the subject of occupational dust inhalation which, in the Province of Nova Scotia is largely confined to coal miners. Of 79 patients in our present study giving coal mining as their occupation, 49 had pulmonary tuberculosis. Of the 30 non-tuberculous, 13 showed definite anthracosis changes in the chest roentgenogram. That the remaining 17 showed no roentgenographic picture of anthracosis we attribute to the fact that most of these patients were not old enough to have been long engaged in the industry, and, during the war were for a considerable time absent from it.

In the cases showing a definite picture of anthracosis, that is an increased hilus shadow, increased linear markings, and particularly a widely disseminated mottling or stippling in the lower two-thirds of the lung, accompanied by a fine reticulation of the peripheral chest field, there was a history of from fifteen to twenty years' exposure in the mines. In these cases we have not seen marked fibrosis or marked dust deposits, such as we have seen in silicosis. In passing we may say that, apart from the occupational history, the x-ray findings were almost the sole ground for our diagnosis.

Of the group making up this study, 238 or 19.8% reported having had definite hemoptysis. Of these, 184 or 77.3% were proved to have pulmonary tuberculosis. 91% of the 184 showed definite parenchymatous changes by the roentgenogram. In the majority of cases having hemorrhages of small or moderate amount, we have not seen parenchymatous change which could be attributed to the hemoptysis per se, but in a few instances we have seen such changes, particularly in the lower third of the lung which might represent post-hemorrhagic infection. Following large hemoptyses, fine parenchymatous changes of a broncho-pneumonic nature and linear in distribution which were probably due to aspiration of a quantity of blood, have been noted in the lower third of the chest, and in some cases widely disseminated from apex to base.

With reference to the x-ray determination of clinical activity, a point which we must attempt to determine on admission and particularly on readmission, we have found that in admission cases a diag-

nosis of activity may fairly readily be made, simply because these patients are coming to us for the first time, and because the recent clinically active case usually gives its corresponding roentgenographic findings, that is, the "fluffy" broncho-pneumonic areas generally seen these cases. However, in the chronic case, in which the disease is apparently stationery, it has been our experience that it is practically impossible to differentiate between active and non-active cases even when serial plates are taken. But in chronic slightly progressive cases usually afebrile, the x-ray has been of great value as the serial roentgenogram may demonstrate the invasion of new fields before there is any clinical evidence of the spread of the disease. Conversely, in retrogressive cases the x-ray has been most helpful in determining clearing and organization and in showing fibrotic changes.

In common with other clinicians, we have found that, while roentgenology is of considerable assistance in differentiating respiratory conditions in simulating the extent and location of tuberculous disease in the lungs. In dealing with the tuberculous war veterans, because so much depends on our examination findings, we certainly would not dispense with roentgenographic study for deciding doubtful cases, many of which, being bronchitic, present a clinical picture closely resembling that of tuberculosis, or in tuberculous cases estimating the degree of disability and in deciding as to the arrestment or cure of the disease, especially in the face of the positive history which is so uniformly and persistently present in many of these cases even when arrested or cured. We could hardly venture to speak authoritatively on the strength of either physical findings or x-ray findings alone. But we think it is idle to speculate upon the question whether x-ray or physical findings should be the deciding factor in diagnosis of doubtful and difficult cases. In the final analysis it must be left to the experience and skill of the physician to combine the two.

If these seem but few lessons to have learned from so extensive a study, I should explain that this is only a preliminary view of our material and that we hope to publish a full statistical report of our findings when we have completed the study of our collected material, which will cover some two thousand cases.

DISCUSSION.

Dr. Dunham: I would like to call attention to a particular location of tuberculosis which does not cause much fever. That is a cavity in the apex of one of the lower lobes. Physical examination reveals no sign of cavity, but we find them by x-ray. The point that Dr. Miller brought out, the character of that lesion, sometimes a small area will be much more vicious than a large area, especially when the small area is breaking down rapidly.

Dr. A. F. Miller: In reply to Dr.——(regarding laboratory method of sputum examination). "We use the direct smear method in all cases as a routine procedure. If sputa are negative for tubercle bacilli after the third specimen has been examined, the concentration method is tried. We did carry on some experiments in culture media, and a few inoculations of guinea pigs. but these are not routine, and are only tried in special cases."

In reply to Dr. Dunham. "I agree with Dr. Dunham that great care must be used in coming to a diagnosis in cases where the x-ray shows parenchymatous changes in the lungs within the 3rd and 4th intercostal spaces. In the few cases of this type in which we have made a definite diagnosis of tuberculosis, it has been usually because tubercle bacilli were present in the sputum. However, several such cases that we diagnosed as "doubtful" later developed manifest tuberculous disease. Now we usually keep these patients for a couple of months and watch developments.

"Regarding the unreliability of history as a guide in diagnosis in the type of case dealt with in my paper, we find in many cases where definite symptoms are given, even persistently present over a period of years, that neither x-ray nor physical findings are such as to lead us to suspect active or clinical pulmonary tuberculosis. In some—I may say in most—of these cases, there is some chronic non-tuberculosis respiratory disease."

X-RAY OBSERVATIONS OF THE PATHOGENESIS OF PULMONARY TUBERCULOSIS

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The purpose of this paper is to outline the knowledge necessary to an understanding of the pathegenesis and progress of tuberculosis of which the x-ray chest plate is a record.

The ultimate aim of all medical research is benefit to the patient. Frequently the mass of detail of scientific study submerges this in the immediate problems to be solved. The test tube and the experimental animal are at our command and in them we are tempted to see end results. Our greatest knowledge of the pathogenesis and pathology of pulmonary tuberculosis has come from two sources; post-mortem studies and animal experimentation.

The most instructive study has been the artificial production of the disease in the animal. In our human patients we have been able to study the actual changes wrought in the lungs by the tubercle bacillus only by the indirect signs elicted by the clinical examination of the patient. We have realized our short comings and errors in translating physical signs into pathological changes within the lungs. Therefore we have seized upon animal experimentation to teach us more, since here we can control our material and make direct observations of the lungs at will.

In our zeal to find out more and more we have pushed our studies on the amimal to such a stage as to provoke the following remark from a learned scientific clinician, "It is almost as important to make observations on the human as it is to make them on experimental animals." We have learned and are learning much from the observation of animals; it behooves us as well to view each human patient as a source of scientific knowledge. Armed with the knowledge gained from experimental studies, the pathologist lays bare the dead lungs on the autopsy table and tells us what has happened. Armed with the same knowledge it is possible for the trained

observer to lay bare the living lungs on the x-ray plate and tell us what has happened, what is happening and what probably will happen. The use of the x-ray plate in the diagnosis of pulmonary tuberculosis is firmly established. Its value does not stop there, for it gives us the means of studying the disease, as a scientific problem in its own unfortunate living host. Anatomy was taught us by dissection of the dead; now in our modern progressive anatomic laboratories the x-ray presents to the student that study in the living. Lung pathology was also taught to us by dissection of the dead, but now the progressive investigator is viewing his post-mortem findings largely as confirmation of his observations and predictions made from an x-ray plate. Such mental dissection of lung lesions from the x-ray plate offer much for our further studies of the disease.

The pathogenesis of pulmonary tuberculosis is explained with wide divergence of opinion, inviting discussion from several angles. The portal of entry of the bacillus which produces the disease in the body is a much debated question. Animal experimentation has failed to settle it to the satisfaction of all. In the experimental production of pulmonary tuberculosis a definite limitation must be recognized. The lesions as we recognize them in the infant can readily be produced in the animal, but the adult type of lesions with cavity formation is rarely, if ever, produced by any of the usual methods of animal inoculation. To produce cavernous pulmonary tuberculosis, whether by inhalation or by feeding, it is necessary to adopt a scheme of introduction of the bacilli whereby they are made to gain entrance into the lymph passages in relatively small doses. When live tubercle bacilli are first introduced into an animal, even if the lung is the primary site of inoculation, a lesion is produced which has little resemblance to the lesion of adult pulmonary tuberculosis. With the first or primary injection of live tubercle bacilli in the animal there will immediately result a relatively slight lesion at the point of inoculation, but the nearest regional lymph nodes will enlarge and show marked reaction. No exudation will occur at the point of inoculation unless the dosage be large. The lesion formed at the point of inoculation will differ according to the dosage administered. With small dosage there will be a slow development of the lesion by proliferation and increase of cells of the fixed type

or the so-called epitheloid cells. With large dosage there will be produced an immediate outpouring of polymorphonuclear leucocytes which rapidly disappear and then the proliferation of fixed cells goes on in an orderly slow manner. When the animal is again inoculated with live tubercle bacilli we observe "Kochs phenomena." There is an active reaction with exudation and necrosis at the site of the second inoculation. This lesion does not progress in an orderly sluggish manner; it tends to heal and no tuberculosis of the regional lymph nodes is produced. So too, if live tubercle bacilli are injected into the pleural cavity of an animal which has received no previous inoculations, no fluid will be produced. But if the inoculation is made into the pleural cavity of an animal suffering with tuberculosis a clear fluid will invariably result. An understanding of these simple experimental observations is necessary to our comprehension of the pathogenesis of tuberculosis in the human. The pathology of pulmonary tuberculosis presents the greatest diversity and multiplicity of lesions. In explaining our physical signs and especially in interpreting our x-ray plates many investigators are too apt to think only of the tubercle as the pathological entity of tuberculosis. We must remember that tuberculosis can mean anything from a small nodule made up microscopically of giant cell, caseation, epitheloid and lymphoid cells, to a whole lung which has become solid, or a lobe entirely honey-combed by cavities. The tissue changes of pulmonary tuberculosis involve exudation, proliferation, caseation, degeneration, necrosis, cavitation, liquefaction and repair. In attempting to explain why the pathological changes produced by the tubercle bacillus in different human hosts can be so extremely varied, many theories have been given and conditions pictured. The virulence of different strains of organism has received exaggerated emphasis. Chemists and bacteriologists would have us take the extreme view of the important part played by colloid chemistry and acidosis.

The normal anatomy and physiology of all individuals at the time of infection gives partial explanation of the pathological changes following that infection, and this is added to by the more abstract reactions of immunity, allergy and resistance. The problem of the unstaple lesions probably will be solved by an understanding of the anatomic defenses always present in the host, and of the intrinsic bodily reactions and extrinsic reactions of the organism.

The anatomic factors of defense are absent in each individual, regardless of the presence or absence of infection. The first obstacle met by any foreign material seeking entrance into the body by inhalation is the fine hair at the entrance of the nose. Beyond this lies the mucous membrane, always moist with secretions, possessed of more or less bacteriocidal action. This membrane lines the nose, mouth and pharynx. In the trachea the ciliated epithelium is so active and capable, that it keeps from the lungs a large amount of the dirt inhaled. No doubt this active waving surface prevents to a great extent, the implantation or entrance of inert dirt or bacteria. This ciliated epithelium lines the trachea and bronchii from the larynx until bronchii of about 1 millimeter are reached[5]. At this point, usually the respiratory bronchus, there is a transition from columnar to cuboidal epithelium and the cilia are lost. Beyond in the ductulous alveolaris and atria the cuboidal epithelium is gradually flattened into the flat single layer epithlium which is continuous throughout the air sacs and alveoli. From the very beginning of the mucous membrane in the nose to the lining of the bronchi in the lungs we find a rich supply of lymph vessels and lymphoid tissue. Dirt accidentally drawn onto the nasal mucous membrane or India Ink injected there experimetally is rapidly expelled or carried into the large lymph nodes of the cervical chain. There is normally no direct flow of lymph from the nasal cavity, pharynx, tonsils or trachea, into the lungs. About the bifurcation of the trachea there is located the large mass of tracheo-broncheal lymph nodes which in our autopsies are found to be heavily pigmented.

Within the inner third of the lung actual lymph nodes with germ centers are found in the walls of the bronchi and particularly at the bifurcation of the bronchi. Beyond the inner third of the lung we continue to find collections of lymphoid tissue, but without germ centers along the bronchi, especially at the bifurcation. In the lower lobes however, the larger lymph nodes with germ centers are found within the parenchyma farther out than the inner third. These masses of lymphoid tissue continue to grow smaller in the finer divisions of the bronchi and vessels and finally are found only at the bifurcations. The end of the ductulus alveolaris is the furthest point along the finer bronchi where we find lymphoid tissue. As the masses

of lymphoid tissue are only filters for the lymph we naturally find lymph vessels in the walls of the bronchi out as far as we find lymphoid tissue. Lymphatic vessels are also associated with the arteries and veins. The pleura has an extremely rich supply of lymph vessels. The lymph flow and the lymphoid tissue within the parenchyma of the lung stand as strong barriers of defense to the invasion of inert foreign particles or living organisms. But beyond the end of the ductulus alveolaris there are no lymph vessels, there is no lymph flow, there is no lymphoid tissue. But the atria, air sacs and alveoli are not left without defense against invasion. Here the phagocyte plays the part of the scavenger in picking up dirt and bacteria, which have penetrated to these depths; carrying them over to the collections of lymphoid tissue where they are then within the realm of lymphatic defense. The role of the phagocyte has been shown to be peculiarly that of a passive barrier. The microscopic study of any lung showing anthracosis reveals these wandering cells engorged with pigment, apparently on their way to the depot of refuse in the lymphoid tissue. Just as pigment is handled so too the tubercle bacillus is disposed of. Excellent studies by Dr. Gerald Webb[20] and co-workers have indicated that while the phagocyte does engulf the tubercle bacillus, it is totally unable to combat or destroy it. Apparently the function of the phagocyte is only to seize and carry the bacillus over into the lymphoid tissue, which possesses the inherent ability to combat the invader. The normal lymph flow in the lung is from the parenchyma toward the hilum, except for a narrow strip of lung tissue immediately under the pleura which can drain into the pleura. The flow in the pleura is over the surface of the lung to the lymph nodes at the hilum. The direction of the flow is established and guarded by valves, which near the hilum open toward the hilum and near the junction of the septa and pleura, open toward the pleura. Pigment is not carried from the hilum into the lung, but vice versa; the tubercle bacillus is not carried from the hilum into the lung, but vice versa; disease does not progress from the hilum into the lung, but vice versa.

Lymph flow, like venous blood flow, throughout the body is maintained not so much by differences in pressure at the source and mouth of the channel, as by constant massage of the vessels by sur-

rounding tissues. The lymph flow is directed by valves as is the venous flow. The venous return from the great toe to the heart is not propelled nearly so much by venous pressure as by the constant massage of the veins by muscular tonecity and active muscular movements of the dependent limbs. Lymph flow within the lung is maintained chiefly by the massage of the vessels due to the reguarly repeated movements of inspiration and expiration and the inherent elasticity of the lung tissue. Promote greater expansion and contraction of the lung and you increase lymph flow. The apices of the lung cramped into the cupolas of the pleura cavities and strapped down by the small circle of the horizontal first rib, undergo a very limited movement. The massage of the lymph vessels is poor, the lymph flow is sluggish and the lodgment of invading tubercle bacilli is comparatively little disturbed. But on the other hand the posterior diaphragmatic borders of the lower lobes experience constant great expansion and freedom of movement; the lymph flow is free and lodgment of tuberculous infection is desperately fought. The record of this phenomena is constantly registered on autopsy findings and x-ray chest plates. This is usually the last part of a lung to become infiltrated by tubercles.

Without an accurate understanding of these anatomic defenses it is impossible to conceive the process of infection, the beginning and dissemination of tuberculosis in the lung. In the vast majority of cases of pulmonary tuberculosis the bacillus enters the lungs by inhalation. It is carried into the small bronchi beyond the ciliated epithelium, beyond the lymphatics and lymphoid tissue, out into the divisions of the primary lobule. There it is taken up by the large phagocytes, just as pigment is handled, and carried to the lymphoid tissue. In this mission the phagocyte acts only as a passive carrier. It does not posses the ability to attack or destroy the tubercle bacillus as I have said, but simply bears the invading germ to the lymphoid tissue where resistance is found. Here in the lymphoid tissue destruction of the bacillus is accomplished or pulmonary tuberculosis begins.

It is not our intention to state that a tuberculous lesion can never start in a capillary or within the lumen of the bronchus. Lesions have been proven to exist in each of these locations but all of our

researches tend to show that whether the entrance of the germ into the lung is by the bronchus or by a blood vessel, the common occurrence is that the bacillus is carried outside of the vessel or bronchus to find lodgment in the lymphoid tissue. This is also the observations of Opie[1] and Krause[4]. Obviously an infection within either a vessel or bronchus so great that the local lymphatics are overwhelmed, results in a lesion at that point. Such lesions occur mostly as the result of experimentation or as late secondary lesions such as caseous broncho pneumonia. Aufrecht[6] and Kretz[7] believed that the lesion starts in the blood vessels. Aufredcht studied the larger veins by histological sections and Kretz used injections of tubercle becilli into the circulation of animals. If Aufrecht had studied serial microscopic sections he would have seen that the lesion started in the lymphoid tissue outside of the vein and extended into the vessel rather than starting from the endothelial cells. Kretz injected such large numbers of bacilli into the blood vessels as to cause emboli in the pulmonary capillaries. The mistake of Kretz has been clearly shown by the careful animal injection experiments of Krause.

With the implantation of the tubercle bacillus in the tissues of the body the pathological changes which immediately result vary with every individual. The lesions of pulmonary tuberculosis are extremely varied, and comprehend practically every fundamental pathological process described. But most striking in the difference of reactions are the changes wrought in the adult and the child. At the very beginning of our researches the great differences between the x-ray chest plates of the tuberculous child and the tuberculous adult were observed and described. It was a great satisfaction when in 1917 Opie[1] discussed these differences from the viewpoint of pathology and post-mortem findings, stressing the points of hypersensitiveness and immunity. Since then a mass of evidence has been accumulated to show that the differences observed between the child and adult are but the examples of reactions in a non-sensitized and sensitized animal.

It has been repeatedly proven and generally known that the first subcutaneous injections of the tubercle bacillus in an animal cause only a slight lesion at the point of inoculation. Dissemination occurs by way of the lymphatics and the nearest lymph nodes undergo a

violent reaction which may progress even to the point of suppuration and discharge. With a subsequent inoculation into the same animal, as when an animal suffering with tuberculosis is re-inoculated, there is an active reaction with necrosis at the site of the second inoculation, and frequently a reaction will occur at the point of first inoculation, which is much more severe than that caused by the first injection. This second lesion is not progressive, it tends to heal and no tuberculosis of the regional lymph nodes is produced. This is known as "Kochs phenomena."

Pulmonary tuberculosis of early childhood does not affect one part of the lung oftener than another. In the first years of life there is almost no tendency for the lesion to heal but progressive caseation occurs; cavities are uncommon but do occur; dissemination readily occurs and miliary tuberculosis is a frequent termination[19]. The lymph nodes at the hilum are invariably markedly involved and usually are the earliest and chief center of the disease. Jordan in examining the chests of children roentgenographically, found these earliest lesions at the hilum. By his pathological researches he proved that the nodes are much more extensively involved than the parenchyma of the lungs and that there is a peribronchial inflammation. Jordan believes in infections of the lungs from the hilum. Ghon[3] recognized pathologically all that Jordan had seen on the x-ray plate and conclusively showed that tuberculosis of the tracheo-bronchial lymph nodes does not occur as the primary focus but is so frequently associated with a lesion in the lung substance that we may assume the invariable presence of the parenchymal focus. Our x-ray observations and pathological studies confirm Ghon's conclusions. The primary intra pulmonary lesion plus that produced in the hilum nodes has been termed the "primary complex" by Ranke. Ziegler[11] recognized exactly the primary seat of the tuberculous focus and describes it to be where we know lymphoid tissue normally exists, but he failed to emphasize the importance of the lymphoid tissue.

Hilum tuberculosis in children with the old Ghon in the parenchyma has all the characteristics of first infection. Following the establishment of this primary complex the disease especially in the young, may become generalized by the blood and terminate in menin-

gitis. With increasing age, however, there is a marked tendency for this primary stage to heal. Various stages in the process of healing may be seen in the lungs of children who die from other causes. These lesions may be recognized as: 1. small caseous nodules encapsulated by fibrous tissue; 2. caseation encapsulated by calcium; 3. calcificaiton with fibrosis. Opie[1] has published some most interesting findings in post-mortem studies of St. Louis children. His figures for the presence of these old lesions found in the lungs of patients dying from causes other than tuberculosis are as follows:

2-5 years;	42.8%	18-30 years;	83.3%
5-10 years;	45.5%	30-35 years;	91.3%
10-18 years;	55%	50-70 years;	93.3%

Examination of the lung on post-mortem may disclose only a few calcified nodules whereas x-ray plates of the chest or lungs clearly show their distribution and number. The routine followed in our studies of correlation of x-ray densities and tuberculosis pathological changes in the lung, was to remove the lungs from the body, inflate them to their normal size and make stereoscopic x-ray plates. From the plates we found we could invariably diagnose and localize such healed areas of calcification, caseation and fibrosis, as to enable us to cut down on them with ease. Just as we localized them with accuracy in the lungs removed from the body, so it is possible to recognize them and place them in the chest in relation to the trunks.

The early puerile lesion as seen upon an x-ray plate consists of a small circumscribed area of density connected to the hilum by a heavy trunk. Such parenchymal foci are usually seen within the mid lung zone. They may be distributed anywhere throughout all lobes and only exceptionally are they found at the apices. There is no special susceptibility at the apices for the primary childhood infection of tuberculosis. The hilum shadows are heavy, showing densities suggestive of calcification, caseation or fibrosis. Sometimes enlarged pulmonary or bronchial nodes may be seen.

The clinical significance of such findings on the x-ray plate of a child is a question of such import as to have caused the formation of a special investigating committee[21] by the National Tuberculosis Association to prosecute such research as would tend to standardize

our interpretations. We cannot decide from an x-ray plate alone when these densities are of clinical significance. Thickened trunks and enlarged hilum shadows on an x-ray plate may be caused by various other childhood diseases, such as measles, whooping cough or mumps. They can never be considered as being the result of tuberculosis unless calcification or caseation are definitely shown to be present. Even then these markings cannot be interpreted as being evidence of active disease unless a definite parenchymal "fan" is visible. But there is one peculiar density seen in the hilum which has clinical significance. This we term a caseo-calcareous node. Upon the x-ray plate it has the density of calcium. It is usually quite large and can be seen to be made up of many small flakes of calcium rather than one stone. Upon microscopic section we find that such lesions consist of a soft caseous center containing calcium and larger hard flakes of calcium within the periphery. Such lesions we have never found to be healed on post-mortem studies and they should always be considered as potential foci of infection.

The report of the special investigating committee[21] includes the following paragraph: "It was the concensus of opinion that children are probably more apt than adults to show definite x-ray evidences in the hilum and trunk shadows of simple as well as serious respiratory infections. Practically all children of the ages of those examined have had at one time or another, one or more respiratory infections, especially measles and whooping cough, that are likely to produce very apparent changes in the shadows mentioned and which will remain distinctly visible for a variable period of time. These apparent deviations from the normal are not necessarily abnormal when observed, but may be the harmless results of one or more infections. No doubt such appearances have many times been misinterpreted as evidences of tuberculosis."

The report in its conclusion states, "Calcified nodes at the root of the lung, without evidence of lung disease, are of no significance except as evidence of some healed inflammatory condition, possibly, but not necessarily, tuberculous. They are a common finding in normal chests". Opie[1] is of the opinion that calcification in the lung or hilum signifies a previous tuberculous infection. We are inclined to accept his views in our studies. The clinical significance of these

markings at the hilum is a question only to be answered by careful physical examination, x-ray studies, laboratory tests and observation. Calcification as seen in the hilum on an x-ray plate may clinically mean nothing. Lesions due to infections which have healed are probably our best protection against pulmonary tuberculosis. However, in order to protect as fully as possible the young patient in his fight for health and to guard the doctor in his diagnosis, we are appealing for the term "Potentially Tuberculous." Any child showing ill-nourishment and under weight, with a history of possible exposure to tuberculosis, running a persistent unexplained fever, giving a positive tuberculin test and showing an enlarged hilum shadow on the x-ray plate, should be diagnosed "Potentially Tuberculous" and treated with the same care as a case of active incipient pulmonary tuberculosis until the symptoms are relieved and the ill-nourishment corrected or another diagnosis proven. In this way in nearly every such instance a frank case of active pulmonary tuberculosis will be averted without stigmatizing the child. In such cases the child has withstood the primary infection and as a sensitized individual is better armed to meet the repeated infections of adult life. The scars in the lungs and hilum nodes stand as evidence of the old infections and speak for resistance gained. This truth was suggested early in our investigations when the first fifty sets of x-ray chest plates of healthy adults showed much greater amounts of calcification within the hilum and much more thickening and studding of the trunks than did fifty cases of active pulmonary tuberculosis.

Adult apical tuberculosis resembles in all respects the reaction to the second infection in experimental animals. It is the reaction of an infection in a sensitized animal. The great difference of reactions to the infection of tuberculosis in sensitized and non-sensitized animals has been shown clearly by many workers[1,4]. At the very beginning of our x-ray studies of the chest the great difference between the plates of tuberculous children and tuberculous adults was noted. The usual case of active pulmonary tuberculosis in the adult is the result of repeated infections. The immunity and resistance gained by the infections of childhood alter the course and picture of the disease in later life. The focal lesions of early life which have healed are beneficial but they do not always effectively protect.

The patient has relative immunity but a diminution of resistance is brought about by intercurrent disease, fatigue, malnutrition and various other factors. Immunity diminishes when recovery occurs and disappears after the lesion is completely healed, but is increased by re-infection, spread or metastasis of the active disease. Reinfection may occur after long periods of quiescience. This undoubtedly happens in human beings, when immunity is comparatively low. So, too, in the adult human infection we find the prompt response of local reaction at the point of inoculation, and the constitutional changes so charastic of the reaction in the sensitized experimental animal.

Before a child is ten years of age he is usually sensitized and has acquired a degree of immunity. Infection after this age produces the picture of adult tuberculosis with apical localization, tendency to chronicity with fibrosis, cavity formation and noticeable absence of associated tuberculosis in the regional lymph nodes. The tubercle bacillus has gained entrance by inhalation, penetrated the epithelial lining of the lung, gained entrance into the tissues where it has been gathered up by the lymphatics and then incited a local reaction in an allergic soil. The first reaction to the implantation of the tubercle bacillus in an allergic soil is an exudate at the point of inoculation. This phenomena is the most striking difference between adult and puerile types of tuberculosis. Peculiarly the exudation takes place immediately under the pleura. This observation has been made by Gardner[18] in his experimental work which I will cite, "With inhalation well developed tubercles appear in about two weeks in a group of alveoli immediately beneath the pleura. Occasionaly in certain animals a few similar tubercles may be found in the mid zone regions, half way from the pleura to the hilum. From these foci bacilli are carried back by lymphatic vessels to nodes at the hilum." Our x-ray observations show invariably that the adult lesion starts immediately under the pleura. This phenomena is vitally important because it explains why we find physical signs early in adult tuberculosis and answers the question why the x-ray plate lends itself so readily to the study of the pathological changes in this type of the disease. We do not attempt to answer why the exudation appears immediately under the pleura. Certainly the lymph flow must play a great part.

Krause[4] has approached this same subject when he asks the questions, "Does human tuberculosis in early life tend to localize at the roots because then the drainage is more active and open? How much inhalation of foreign bodies and respiratory infections alter the puerile lymphatic structure?" The blocking of the lymph flow toward the hilum is clearly shown in studies of tuberculous lungs. The engorgement of the peripheral lymphatics may be brought about either by a blocking of the central lymphatics or by an overflow of exudate. It is a frequent observation to find dilated pleural lymphatic vessels over a lesion within the lung and on section find that pressure on the lymphatic vessels running toward the hilum has caused the lymph flow to seek out the collateral route toward the pleura. This is made possible because of the position of the valves. The normal lymph flow through the lung, which occurs from the parenchyma toward the hilum, except for a small area which is immediately under the pleura and which may drain into the pleural lymphatics, can be seen to have a vital bearing on the pathology and progress of the disease.

As before stated an exudate is the first reaction to the implantation bacilli in the tissues of a sensitized individual. If a massive dose has gained entrance at any certain point the exudation is of the polymorphonuclear type, due undoubtedly to the reaction of tissue to a foreign protein. But this polymorphonuclear exudate disappears in about forty-eight hours to be replaced by a mononuclear exudate and proliferation of fixed tissue cells. From these elements the characteristic microscopic picture of tubercle formation with giant cells, epitheloid cells and lymphocytes is produced.

From this point the pathology, progresses either to the reparative changes of fibrosis and calcification or the destructive changes of caseation, ulcerataion and cavitation. When an adult individual is in possession of the ordinary amount of allergy and resistance, any lesion of pulmonary tuberculosis always tends toward healing. It is also true that the lesions of an adult type of the disease are the results of repeated infections either from without or within. For instance the first lesion within the lung of an individual case, let us say, is in the right apex. This particular lesion has gone through the pathological changes of exudation, tubercle formation

and is now present only as an old fibrotic scar. But while this oldest lesion has been progressing to repair and healing, let us say a newer lesion is developed at some other point lower down in the lung or in the opposite apex. This newer lesion may be the result of spread from the original lesion or a new infection from without. This second lesion we will say is in the state of exudation. Drawing the picture still farther, we may have developed an area of caseous broncho pneumonia in one of the lower lobes. This lesion is the result of aspiration of infectious material from an apical lesion. Now in this problematical, yet typical case there are three distinct lung lesions, all due to tuberculosis and each one in a different state of pathological change. There is present exudation, caseation and fibrosis.

We must turn again to anatomy to explain these lesions as we find them on physical examination, on the x-ray plate and in post-mortem studies. Septa are prolongations of the connective tissue from the pleura down into the lung. These connective tissue septa, rich in lymphatics act as excellent barriers to the spread by continuity, of any lung pathology characterized by exudation. Inflammatory exudation starting within any one of the small compartments, anatomically known as "secondary lobules" is definitely limited by these barriers. Spread through such a septum will not take place until cavitation occurs. The localization and restriction of the pathological changes by these septa accounts for our localized rales on physical examination and also explains the "Fan"[15] on an x-ray plate.

Let me state, however, that these fans may be present in any lung disease producing exudation. The cellular elements of an acute lobular pneumonia, apical catarrh or an infarct are all held in check by the septa and can consequently give localized signs and a "Fan". The differentiation of these lesions can be made by an understanding of the progressive pathology of tuberculosis just described and the ability to recognize it upon an x-ray plate.

From extensive research it has been established that the densities caused by the pathological changes of pulmonary tuberculosis as shown on an x-ray plate vary in their quality and degree of density, progressing from the least dense to the heaviest as follows:

1. serous exudate, 2. cellular exudate, 3. fibrosis, 4. caseation, 5. calcification. Given then, the problematic case cited above or any case of adult tuberculosis showing two or more lesions, the x-ray picture will be characterized by the densities of different qualities. A thorough study of x-ray chest plates with this understanding will enable you to read the changes in terms of actual pathology. The localized, cone-shaped lesions, walled in by the septa will never be seen in post mortem studies if the pathologist's usual sweeping cut from apex to base is made. But if you will carefully dissect out along the bronchi you will easily lay bare these lesions immediately under the pleura. So, too, if you read your x-ray chest plates with no respect to trunks you will never see the fan. But again if you will follow out along the different trunks, the lesions present will not appear as one continuous mass of pathology but you will be able to mentally dissect the changes and translate them in their actual progressive pathology.

After the apical lesion, the most common lesion of adult pulmonary tuberculosis is caseous broncho pneumonia. It is invariably associated with old apical lesions, usually containing cavities. It invades the lower and middle lobes and lower part of the upper lobes and not infrequently the apex opposite to an old lesion. It is in all probability caused by aspiration of sputum droplets containing large numbers of the bacilli and so can be described as the reaction of massive infection in a highly allergic soil. Upon gross section of the lung and microscopic studies it is recognized as diffusely scattered yellow nodules of caseation, between which lie areas of normal lung tissue. Upon the x-ray plate it is easily recognized as a diffuse heavy mottling, frequently seen scattered along the main trunks suggesting "raisins upon a stem." It may become confluent and produce pseudo-lobar caseous pneumonia. As confluent localized areas it may take on a "Fan" shape. Caseous broncho pneumonia may exist in a large area without causing any signs or symptoms. Its recognition in any case has always led us to a grave prognosis. Recent studies however, have made radical changes in our views relative to the clinical gravity of this complication. The observations which have startled us and made us shift our views is the extent to which caseous broncho pneumonia can be absorbed. Too often has our

grave prognosis, given because of the presence of caseous broncho pneumonia, been mocked by the clinical cure of the patient and evident absorption of the exudate.

The possibility of the absorption of tuberculous exudate was given consideration by early workers. Ziegler[11] states: "It is very doubtful whether complete recovery of the effected tissue by reabsorption of the exudate is in any case possible, and indeed it can only occur in the very smallest patches whose vessels are not yet obliterated. In larger patches, healing can only take place when the inflammatory process issues in fibrous hyperplasis and induration." However, Virchow[10] in 1858 stated.: "These cheesy transformation is the regular termination of the tubercle, but on one hand, it is not the necessary one, in as much as there are rare cases in which tubercles in consequence of their undergoing a complete fatty metamorphosis, become capable of reabsorption; on the other hand the same cheesy metamorphosis befalls other kinds of cellular new formation, for pus may become cheesy and likewise cancer and sarcoma."

Recently Gardner[18] in his excellent studies on the healing by resolution in experimental tuberculosis has shown conclusively that "caseous tubercle can heal by resolution and finally disappear completely." The interest in these experimental studies is manifoldly increased by the work of Dunham and Norton[17] on the x-ray studies of the absorption of tuberculous exudate within the lung. The latter report noticeable absorption of tuberculous exudate in 59 cases of a series of 489 or more than 12 present. In 7 cases the change was slight, in 24 the change was moderate, in 18 the change was marked. In all there was clinical improvement of the patient. Since the report of this series of cases the same observations are constantly being made by us in our office and at the Cincinnati Tuberculosis Sanatorium. Here again the experimental and post-mortem observations of a most vital phase of the disease can readily be followed and studied more fully in the living human. The clinical importance of the scope of this work is tremendous. The observations are startling because the changes noted are neither slight nor infrequent.

In seeking explanation for this phenomena of absorption of tuberculous exudate anatomy may again show us the answer. Miller[5]

in his newest work on reticulum of the lung has shown that reticulum precedes white fibrous tissue in the formation of tubercle and scar. By special stains of very thin microscopic sections the location of tubercle can be detected by the arrangement of reticulum even before the cellular elements of a typical tubercle can be made out. But in sections of caseous pneumonia reticulum is noticeably absent or at least not so prevelant. Reticulum precedes white fibrous tissue. In caseous broncho pneumonia reticulum is comparatively less; therefore may we deduce that it is more possible of absorption?

Lobar or massive caseous pneumonia occurs in both the adult and child. In the adult type old apical lesions are always present. Large areas of the lung may be involved for months without causing severe symptoms. The old apical lesion is seldom found by physical examination and so, although a definite tuberculous lesion may have existed for some time, it is too frequently only recognized when the massive lesion has progressed to cavitation and death is close, thence the name "hasty consumption." Allergy as shown by Krause[4], teaches us that "if a man falls acutely ill with tuberculosis it means that there has been rapid fresh extension of metastasis of infection from pre-existing foci, known or unknown." In x-ray studies the old lung foci are found and the progress of the new massive lesion followed.

Acute miliary tuberculosis deserves special consideration. We believe it is the result of lack or loss of immunity. Krause[4] states "miliary tuberculosis is always the result of re-infection from older foci." Thus we find it associated in the adult with definite old healed lesions as revealed by the x-ray plates. In children the old focus may not be evident, but a Ghon is usually seen or the primary focus found in other parts of the body. Many cases of healed miliary tuberculosis are being found by our more general use of the x-ray chest studies. This diagnosis is only justified when the subject gives no history of work in a dusty trade: Lungs of anthracite miners most nearly simulate miliary tuberculosis. The x-ray picture is that of fine sharp, discrete studdings more or less evenly distributed throughout all lobes. It has been our good fortune to have had two such cases come to autopsy and to be able to definitely prove the diagnosis. Most peculiarly these patients seldom if ever give any

history suggesting serious lung infection. Opie[1] in his pathological studies has noted the same and states, "such extensive calcified tubercles of the lung and its lymph nodes were disclosed, that a grave infection must have existed at some time previous; even though there had been no history of corresponding symptoms, there was doubtless at some period imminent danger of death from tuberculosis. This form of infection has an interest not only for the pathologist, as often assumed but for the clinician and for those interested in the epidemiology of the disease." Such diffuse lesions located by the x-ray chest plate in an individual apparently well, may have no greater clinical significance than that there is definite loss of lung function; even shortness of breath may not result. Furthermore it has been our observation that when a new tuberculous infection is superimposed upon these old healed miliary lesions, the progress of the case is rapid and the prognosis necessarily grave. This was taught us by studies of the living, made with x-ray plates, plus the knowledge given us by the pathologist.

The predominance and significance of caseous pleurisy has been greatly emphasized by x-ray studies. The x-ray picture is that of empyema associated with caseous broncho pneumonia without old apical lesions. At U. S. General Hospital No. 19 we had opportunity to study 17 cases by x-ray plates and careful post-mortems. The pleura shows massive thickening with solid caseation, involving chiefly the parietal pleura. The lesions within the lungs are comparatively slight. Old apical lesions are seldom if ever present. In some cases the solid caseation of the pleura measured 5 c. m. in thickness. The end is invariably rapidly fatal, although the patient neither feels nor looks sick. Literature reveals meagre mention of the condition. Letulle[22] gives a short concise microscopic description with some excellent illustrations. German investigators during the war reported a few cases among the Arabian troops in Turkey. To us caseous pleurisy represents the massive reaction of an individual devoid of resistance. It is comparable to the other massive lesions found in the lymph nodes, or represented by lobar caseous pneumonia as found in the aborigines and other primitive tribes. This has been emphasized by Bushnell[2]. Of our 17 cases studied, 14 were found in young adult negroes coming from rural districts.

The x-ray studies in each case showed the massive changes and absence of old apical lesions. The type of case and progress of the disease was followed in the living, by the x-ray. Our deductions were proven on autopsy.

In conclusion we wish primarily to emphasize the importance of the "fans" or cones which are so constant in and characteristic of the adult apical type of pulmonary tuberculosis, to note their differing densities and their definite location within the lung and to contrast the importance of this finding with the conception of pulmonary tuberculosis as expressed by hilum and per-bronchial tuberculosis.

Hilum and peribronchial tuberculosis are terms born of a false conception of the pathogenesis of pulmonary tuberculosis. It is conceded that the tubercle bacillus enters the system in one of many possible ways, such as passing through the large bronchial walls, to the bronchial nodes or through the intestinal tract without involving the mesentary nodes and in some mysterious way reaching the bronchial nodes. This is supposed to occur usually during childhood. The infection then lies dormant in the bronchial nodes for many years and sometimes after puberty, immunity or resistance is lost and the infection passes backward through lymph strains to the parenchyma of the lung by way of the peribroncheal or perviscular lymphatics. Thus we have first hilum tuberculosis and then paribroncheal tuberculosis and finally parenchymal tuberculosis.

This conception, which seems false to us, received its greatest impetus since the study of x-ray chest plates has become general. It is contrary to our knowledge of anatomy, pathology and to the modern conception of pathogenesis which has been so ably described by Krause under the term of Allergy.

In contrast to this we conceive that the adult and puerile types of pulmonary tuberculosis which are so clearly differentiated by the x-ray chest plates have a firm foundation in anatomy, pathology and allergy. The infection enters the lung by the air passages or the blood stream, is carried from either to the lymphoid tissue which is directly connected with the lymph vessels. In the child we have very little reaction except near the point of inoculation and this is mostly interstitial, but a great reaction of the regional lymph nodes. In the adult we have comparatively little reaction of the lymphnodes but

massive reaction around the infection with extensive exudate which floods a definite part of the lung. Thus in the child which is not sensatized the reaction to the tubercle bacillus is little like an inflammatory reaction, while in the adult the reaction is a severe inflammatory reaction and is seen upon the x-ray plate as a lobular density under the pleura like a fan or cone in shape as it is confined by the lung structure. Pulmonary tuberculosis is a disease of successive infections, therefore of differing ages which causes the fans to have differing densities. These differing densities are easily recognized upon the x-ray plate. The only tuberculous lung lesion which we have observed on the series of x-ray plates of a given patient, that spread from the hilum to the periphery is tuberculosis caseous broncho pneumonia. This spreads by aspiration of droplets from open lesions. This lesion must not be confused with perivascular or peribronchial tuberculosis. It can only originate from the hilum where a tuberculous lymph node ruptures into a bronchus and infectious material is aspirated into the smaller air passages.

We have tried in this paper to review in abstract form the facts and literature which we find necessary to the reading of a chest plate. such readings require more than a knowledge of clinical tuberculosis or roentgenology. They require a knowledge of anatomy, pathology, physiology, pathogenesis of tuberculosis, in addition to clinical tuberculosis and roentgenology. Is it surprising that so many of you depreciated the x-ray readings which you have received and which you have not understood?

REFERENCES

1. Eugene L. Opie. Pthisogenesis and Latent Tuberculous Infection. Am. Rev. Tbc.—Sept. 1922 Vol. VI. No. 7.

The Focal Pulmonary Tuberculosis of Children and Adults. Jour. Exper. Med. June, 1917.

The Relation of Apical Tuberculosis of Adults to the Focal Tuberculosis of Children. Jour. Exp. Med., Aug., 1917.

First Infection with Tuberculosis by way of the Lungs. Am. Rev. Tbc., Nov., 1920.

2. Bushnell. Epidemiology of Tuberculosis. New York, 1920.

3. Ghon, Anthon. The Primary Lung Focus of Tuberculosis in Children. J. and A. Churchill, London, 1916.

4. KRAUSE, A. K. The Significance of Allergy in Tuberculosis. Trans. of 17th Meeting of the Nat'l Tbc. Association.

Tuberculosis. —Nelson Loose-Leaf Medicine. 1921.

Experimental Studies on Tuberculous Infection. Am. Rev. Tbc., Mar., 1922. Vol. VI, No. 1.

The Nature of Resistance to Tuberculosis. Am. Rev. Tbc., Nov., 1920.

5. MILLER, W. S. Anatomy of Lungs. Reference Handbook of the Medical Sciences Vol. 6; 1916. Wm. Wood, N. Y.

Some Essential Points in the Anatomy of the Lungs. Am. Jour. Roent., June 1917.

Reticulum—Work unpublished.

6. AUFRECHT. Der Gegnenwartige Stand dir Lungen—schwindsuchfroge. Berl. klin Wochenscha, 1907.

7. KRETZ. Uber Pthesiogenes, Bertr. z Klin d. Tuberk. Vol. XII, III.

8. NICHOL. Die Entwickelung and Einteclung der Lungenpthise. Bertrage zur Klinik der Tuberk. Vol. XXX, No. 11.

9. TENDELOO. Studien uber die Ursachen der lungenkrankherten. Wiesbaden, 1902.

10. VIRCHOW. Die Krankhaften Geschwultse, 11.

11. ZIEGLER, ERNST. Pathological Anatomy and Pathogenesis. Wm. Wood and Co., New York, 1887.

12. WERGERT. Virch. Arch. Vol. 77 No. 87.

13. ABRIKOSOFF. Uber die Ernstin Anatomischin, Veranderungen der Lungenpthese. Virch. Arch. 178.

14. BUSCH, HIRSCHFELD, F. V. Concerning the Position and the development of Lung Tuberculosis. Deut. Archiv. fur klin. Med. 1899, Vol. 64.

15. DUNHAM, KENNON. The Relation of the Pathology of Pulmonary Tuberculosis to the Roentgen Findings. Am. J. Roent., June 1917.

The Cone and Collateral Lymphatic Circulation in Pulmonary Tuberculosis. Am. Rev. Tbc., Oct. 1920.

X-ray Chest Manual. Trans. 14th Meeting of Nat'l Tbc. Assoc. Stereoroentgenograph—Southworth Co., N. Y.

Clinical Observations Derived from the Examinations of over three thousand chests checked by Stereoroentgenograms. Am. Tbc., Oct. 1917.

X-ray examination of the Chest and an X-ray Classification of Pulmonary Tuberculosis. Am. Jour. Roentg. Vol. VIII, No. 8.

Localization of Pulmonary Lobes by the X-ray. Med Record., June, 1920.

Localization of Pulmonary Lober by the X-Ray. Med. Record, June, 1920.

A Study of Tuberculous Lung Lesions as Revealed by X-ray plates and the Value of These in Physical Diagnosis., J. Med. Soc. New Jersey, 1917.

16. Dunham, K. and Skavlem, J. H. A comparative Study of the Pathology and X-Ray Densities of Tuberculous Lung Lesions. Am. Rev. Tbc., June 1921.
Sinus Disease and Lung Infections. Jour. of Rad., Feb., 1923.

17. Dunham, K. and Norton, V. An X-ray Study of the Absorption of Tuberculous exudate Within the Lung. Am. Jour. Roent. Feb., 1923

18. Leroy U. Gardner. Healing by Resolution in Experimental Pulmonary Tuberculosis. Am. Rev. Tbc., May 1922, Vol. VI, No. 3.

19. Lawrence W. Smith. Tuberculosis in Infancy and Childhood. Am. Rev. Tbc., May 1922, Vol. VI, No. 3.

20. Webb, G. B., Ryder C. and Olcott, C. The Lymph Node Distribution of Experimental Tuberculosis in the Guinea Pig. Am. Rev. Tbc., Sept. 1920.

21. Committee of National Tuberculosis Association. Clinical and X-ray Findings in the Chest of Normal Children. Am. Rev. Tbc. July, 1922.

22. Letulle, Maurice. La Tuberculose Pleuro-Pulmonaire. A Maloine et Fils. Paris, 1916.

Dr. Dunham: The greatest trouble in using an x-ray plate is when the reader does not know the normal chest plate of an average individual. It is of the greatest importance to be able to positively state by an x-ray reading of the chest plate that the man has no pulmonary tuberculosis. It is also vitally important to be able to state that although there is tuberculosis present, the symptoms of which the patient complains cannot be accounted for by the condition in the chest. When we find rales on physical examination and the x-ray plate does not show a corresponding lesion let me emphasize the importance of a thorough study of the nasal sinuses as a possible source of infection.

I hope you will clearly understand the big point that in the interpretation of x-ray plates a knowledge of tuberculosis in all its angles is necessary. A thorough knowledge of clinical tuberculosis and an understanding of the significance of physical signs is of great importance. Therefore you men who know tuberculosis will best be able to read your own plates. In answer to Dr. Webb's question as to whether we have seen any of these things develop I may state that we have observed children in which at first there was no evidence of pulmonary tuberculosis. They were classed "Potentially Tuberculous" because they came from tuberculous families, were definitely undernourished, some gave positive Von Pirquet reactions. In a few of these cases by observing serial x-ray plates we have seen fans develop.

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